CHEST EXAMINATION

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THE CORRELATION OF PHYSICAL AND X RAY FINDINGS IN DISEASES OF THE LUNG

By

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FOREWORD

WHILE still a medical student I f lt strongly that the next generat on would ondemn the practi f herding cases of proved pulmonary tuberculosis in out patient d partments There they would it n row among the othe patients oughing o them ultimat ly to be rewarded by a bottl of od ll er o l and some linctus But w did ha the pportunity of l arning our phy ical gns from these unf rtunates To-day I can affirm from my xpe ence as an examin r t arious un ers t es that the weakest part of the candidates equipment is the r ecogn tion and int pret tion of physical signs in the lungs T some xtent this may be due t the segreg tion f tuberc lous cases nto departments where students do not freely ent but st ll mo e t the t nden y to short-c rcu t the r xam nation by flying t th X ray film using t as a ubstitut nstead f confirmation f ordinary clinical methods Th t achers however re not entirely free from blame particularly the matter of multiplying labels fo the arious sounds he d through the stethoscope the degree of ac uracy thereby mpl ed is largely illusory and merely onfuses th tudent It is one of the many merits of Wing Commander Tra l book that h f llow the tradition f my own t achers D Gee and D Samuel West by us ng as few t rms as possible and orrelat g ach w th ts pathological significanc

This plan is haract ristic of the whole work anatomy phy io- logy pathology ymptoms physi al gns and X ray findings ar losely orrelated enabling the tude t r practitione to build up ment l concept of exactly what is happening inside the hest Many will be interested to l arn how greatly modern X ray tech- nique has advanc d nt rpretation in k lled hands E mo w ll be surprised t find what h lpful deductions from phy io- logi l principles they hab tually ign re And all w ll be relieved to find how grasp of the first principles inv l ed w ll enable them t rri t rrect opinion w thout mposing burdensome deta ls on th memory

As f rme t ache I know how aluabl a ontribution this book makes t the understanding of tal p blems wh le the freshnes of it approach w ll be patent to ll

W LANGDON BROWN

PREFACE TO THE THIRD EDITION

THIS Third Edition has again been slightly enlarged Reference has been mad to recent work on the broncho-pulmonary segments of the lung a chapter has been added on miliary shadowing and the last chapter of the second ed tion has been replaced by short description of lung tumours

To llustrat these additions xtra figures and X rays ha been included and for som of th X ays he e eproduced I wish to express my gr teful thank t D P t K rley and to Dr Fawcitt, who has don such excellent work on radiological vidence in haematit iron-ore w rk rs

R R TRAIL

PRIFACE TO THE FIRST EDITION

THIS book is founded on courses of lectures which t has been the writ s good f rtune t gi to tudents and post graduates They ow thei present form in great part t attempts made t answer the difficult es f those students who howed an int rest an pproach to exam nation f the chest f unded on know ledge of ppl ed anat my physiology and pathology without whi h t is naturally useless to attempt a reasonable i terpretation nd correlation of phy ical and X ray find gs

C rrelatio is not so d fficult to acquire as many students seem t fear Much confusion has arisen of late y ars because of the b g ad ances mad by hest phy ician specialists on the one hand and hest adiology specialist on the other More recently howev r there has been a tendency to ombine these branches in the hest physi ian cum radi logist r the chest physician who likes to read his ow X ray This is ll t the good of the modern tudent t tends t bring us back to fundamentals We find it increasingly asy t go beyond th objective reading of films W an connect phy ic l igns with definite bnormalities of

shadow and combine both with the changes in normal anatomy and physiology so ably expressed nowadays by the specialized lung pathologist who is ve must remember but the first off hoot of that type of learned *physician who laid his firm foundatio* for practice n his earlier post mortem room researches when X rays did not exist

The first Section of the book is therefore devoted to reminders on those salient points of normal anatomy and physiology hich explain the abnormalities of the commoner chest diseases of general practice Like *other points of equal importance they* are repeated by reference to oth r sections

In the second Section on Applied Pathology we c nsid r the main gross and microscopical changes induced *by these di eases* and in noting the physical signs and the alterations from the normal postero-anterior film that accompany them in their various tages an attempt is made to corr late all three aspects A c rt in amount of detail d description is necessary but this is confined as n uch as possible to fundamentals even if by this staterr nt v appear to be begging th question

There has been of late years much discussion on the reading of *abnormal films and of necessity so much nd vidual ariation* in reports that an attempt has been made in son e quarters to confine th m to objecti e v ording As already indicated th v riter fe is that e can go furth r that we should attempt t arrive at riteria for interpretation using th ork of the phy i ian the pathologist nd th radiologist as a combined whole W shall ll agre that changes of microscopical detail cannot be expect d to reflect themsel s in stethoscopic igns and on the usual post ro-anterio film e know that a l bule on the lung urf f e is no more than about one-quart r f *an inch in its longest* diamet r Nev rthel ss it is felt that a kno l dge of pathogenesis is fundamental for the stud nt who ould orrelate his physical and X ray find ngs e en if these are demonstrable only vh n comparati ely gross areas of lung are i ol ed Thus we know that in phthisis stethoscopic signs r lat but can find a *reason why th y a e lat* and so why we hould look for ther and *as important physical igns such as lack of mo* ment and note that ill preced these stethoscopic signs and so warn *us of the presence of a* pecific pathological change It may be rgued that the prime correlations here detailed re built on *prior* reasoning even if this be *justifiable criticism it will be admitted that they*

can g v some l mentals that may combine the till too-w ll defined mpartments f the patholegist the radiologist and the physi ian I v r a mere workabl planation wo ld be bett r than no basis t ll

Section III sums up the phy i l findings in the diseases discussed n Section II nd gi a scheme f nt rpretation f stethoscopic findings that may act as basis f diagnosis w thout X ray findings N tt mpt is ma l t d al with ll normal and abnormal tethoscopic igns but only w th uch ad entitious sounds s an be c rrelated w th underlying pathogenesis It is taken f r granted that th tudent has listened to large numbers of normal hearts and a ufficient numbe f abnormals t understand the p ir ples und rly ng the int rpretatk n of breath sounds and ce sounds g th diff renc between the prolonged xp ratio of emphysema nd the high p tch faint loud t the a that onnotes bronchial bre th ng

It w ll be seen that all these sections are int -connected They are n essenc th parts of an nteresting jig-saw puzzl True they plac personal lues on the individual piec used in the g me but they re the result of the writ onscious attempt over se ral rs t ncrease th al of each piece by using pe t nc t lt r its hape so that t fits more eas ly w th ts f llow l h new problem n l ing pathogenesis ontinue to be instruct wh l t whet the appet t fo till furthe problems

The ult mat im of this handbook is to bring added help to the tudent ndergr duat and post-graduat in the assessment of the l ing and hanging p thogenes s f his individual patient only by so doing an w rn t r asoned basis f that wider problem f nd idual tre tment which must be the bject and is alway the l true ew rd f the happy phy ician

M thank re due t Si W lt Langdon Brown f r his kind ness n writ ng Foreword t F/Lt J A Kennedy f his assistanc read ng proof and i pr paring the nde and to Mr J Ru rs of Messrs J & A Churchill Ltd f H s valuabl help n these da of d ffic lty with paper and print

R R TRAIL

CONTENTS

SECTION I APPLIED ANATOMY

SECTION II PHYSICAL EXAMINATION

SECTION III THE ABNORMAL FILM

CONTENTS

SECTION I APPLIED ANATOMY

SECTION II PHYSICAL EXAMINATION

SECTION III THE ABNORMAL FILM

CHEST EXAMINATION

SECTION I APPLIED ANATOMY

CHAPTER 1

THE BRONCHIAL TREE

The trachea lies behind the great vessels embedded in the elastic reolar tissue which ensheathes all the structures lying in the mediast num It divides t about the level of the fifth dorsal ertebra nt Its two main branches the right and left bronchi To this point it can be seen on the normal postero-anterior film of the chest as clear area, bearing slightly t the right of the mid line of the thorax

The point of division is important in all cond tions which cause displacement of the mediastinum in that it lies not far below the third dorsal ertebra opposite which is the weakest part of the mediastinum On this point the mediastinum swings laterally as on a hinge Any pull on the lung structures connected with one main bronchus is easily transmit ted along the lower part of th trachea to this w ak point W shall see later that the trachea responds more than any othe mediastinal structure to lung changes, and that therefore if w find vidence of ts disturbance w can reason backwards to find the cause in such lung hanges Fortunately its movement quickly reflected on the st momast id muscle so that we hav an asily demonstrable ign of its displacement

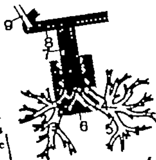


Fig. 1 The Mechanism of the Sternomastoid Sign.

1. The right upper lobe bronchus.
2. The right middle lobe bronchus.
3. The right lower lobe bronchus.
4. The left upper lobe bronchus.
5. The left lower lobe bronchus.
6. The areolar tissue of the mediastinum
7. The pretracheal fascia.
8. The fascia of the neck
9. The sternomastoid muscle.

The anatomical relations of the muscl explain this reaction (see Fig) On the anterior surface of the trachea lies a band of tissue called the pretrache l fascia connected be low with the lastic areolar tissue of the mediastinum, and abov with the

cases of substernal thyroid and in adenoma of one lobe of the gland which pushes the trachea out of its central position.

Along with other mediastinal structures the trachea is displaced in the common abnormality of dorsal scoliosis which is usually convex to the right. Th is really a t rsion of the thoracic cage pushing the right lower half f rwards and outwards and pulling the left lowe half inwards and backwards as viewed anteriorly When we look t the patient from th fro t w see his right lower ribs forced apart to produce bulging whil the left lower half appears t be fallen in xactly the ppoint is found on looking at the back the right low zon is flatter and th left lower zone more prominent than the normal In the w rds th volum of the chest cavity as whole is not reduced The effect of this

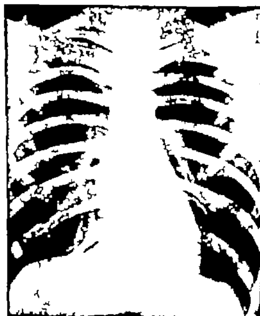


FIG. 2. Normal postero-anterior film.

torsion on the hadow of the heart and of the main vessels will be discussed in the chapters on th mediastinum i health and disease and on the normal postero-anterio film

deep fascia of the neck The latter fascia which meets from both sides in the middle line divides as it goes backward to enclose the sterno-mastoid muscle and is thus in close contact with its tendinous part which has its origin at the anterior superior border of the manubrium sterni We see therefore how it is that tracheal displacement is reflected to an increased tension of the sterno-mastoid muscle in this tendinous part *on the same side as that to* which the mediastinum is pulled or pushed by traction on one side or pressure from the other

As we proceed with our studies we shall find that a pull on the trachea is effected by all processes which interfere with the elastic tissue of the lung This elastic tissue is continuous throughout the bronchial tree and carried from its smallest division to the elastic surround of the air cells indeed with the finest ultimate arterioles it forms the actual alveolar wall

All branches of the bronchial tree are also intimately surrounded by a binding of connective tissue which acts as a supporting struc ture like scaffolding It is resilient and moves in response to movements of the bronchi as they alter in length and diameter by inspiration and expiration but it is not an integral part of the bronchus like the elastic tissue and so its pathological changes have not the same opportunity to reflect themselves on the mediastinum Its changes appear to act only secondarily Its commonest patho-logical change is in the deposition of fibroblasts which produce peribronchial fibrosis Increase in depth of tissue leads only to ultimate shrinkage and loss of resilience what seems to happen is *that the fibrosis obliterates lobules it was meant to support* and causes such a drag on others in its neighbourhood that it destroys their elastic tissue after distending them

Direct and indirect destruction of elastic tissue occurs quite early in adult pulmonary tuberculosis Material from diseased lobules enters and then blocks the supplying terminal bronchiole As air cannot now reach th alveoli empty ones collapse, and full ones organize and both lose their elasticity Th trachea is thus pulled to the diseased side and we find a sharp inner border to the sternomastoid muscle on the same side

The effect of the mediastinal hinge on tracheal displacement is well shown in disease confined to the upper lobes It is not unusual to find that the part above the third dorsal vertebra is dragged into a definite bow by localized infraclavicular tubercu-losis In the same way a kink in this region is seen in several

the upper lobe is supplied by two branches convex outwards that come well down towards the left cardiac border supplying the thin triangular part of the lobe that lies in front of the lower lobe

If any of these normal markings are altered in distribution or have disappeared we can conclud which part of the bronchial tree has been interfered with by disease and so get a very helpful aid to diagnosis.

Thus on the film of right lower lobe pneumonia we see no shadows of the blood supply to the diseased lobe If we can f llow the markings of the rest of the blood supply to the lung we



Fig. 4

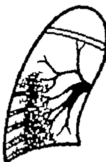


Fig. 5



Fig. 6

shall find them quite normal in thei distribution (see Fig 4)

If th disease is right pleural effusion w can find the shadows of congested blood vessels in the compressed lung, internal to the shadow cast by the fluid There is no obliteration of blood supply (see Fig 5)

On the film of pleural effusion complicating lower lobe pneumonia w shall mak out the shadow of the fluid tow rds the periphery internal t it w see no markings f blood supply in the diseased lobe The rest of the blood supply is normal in distribution (see Fig 6)

If w are dealing with case of collapse of the right lower lobe w shall not two effects on the shadow of the blood supply F rst there no markings in the area of collapse which is more o less opaque and appears t be continuous with the heart hadow Second as the lower lobe has ollapsed and shrunk the mar

It is interesting to note that the effect of scoliosis on rib-spaces is no longer seen when pulmonary tuberculosis supervenes Compensatory emphysema seems to undo it

British and Continental writers give differing descriptions of the bronchial divisions The latter describe the branchings as monopodial coming off what they consider to be the primary bronchus in the continuous stem from the bifurcation to the base of the lung British writers have used the nomenclature of Ewart who described the branchings as dichotomous This is not exactly true because consecutive divisions often follow on each other too closely to demonstrate dichotomy and the individual divisions differ so much in diameter that three or more may seem to arise at one point. We cannot follow these divisions on the chest film, but we can see the main divisions of the vascular supply and these are so similar and so closely approximated to the main bronchial divisions that they form a practical guide to them. They can be traced on a good postero-anterior film throughout three-quarters of the lung fields from their origins in the hilar regions There is one point of difference that is important when reading abnormal films blood vessels continue below the shadow of the diaphragm bronchial shadows do not unless they are enlarged by disease

The diagram of the blood supply shows the branches of the pulmonary arteries as they appear on a good postero-anterior film The right pulmonary artery makes a T-shaped shadow at the hilum It gives off three branches to the upper lobe, one to the middle lobe, and three to the lower lobe The left artery gives an elbow shaped shadow at the left hilum, about half an inch higher than the right one on a 15 × 2 inch film It *gives five b anches to the upper* lobe and four to the lower lobe It will be noted that much of the left lower lobe supply is hidden by the heart shadow and that the lower part of

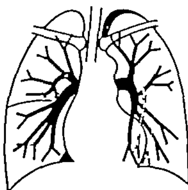


FIG. 3. The Blood Supply The posterior branch of the right upper lobe blood supply and the blood *supply to the lingula of* the left upper lobe are shown in dotted lines.

off-shoots because uch conditions as tuberculosis abscess of lung and collapse may be in an area d limited by the air supply from one particular bronchus This area is cone-shaped th apex of the

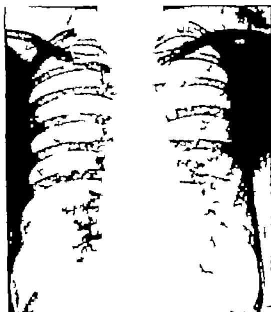


FIG. 9 Congenital cystic disease complicated by collapse and bronchiectasis in the left lower lobe.

cone lying t the entrance f the bronchus This explains why disease of any one such area which lies laterally in th lung will cause triangula loss of translu ency on the postero-anterior film

In 932 Krame and Glass of America introduced th term broncho-pulmonary segment They defined it as the area of parenchyma suppl ed by a constantly placed bronchus wh ch opens off large lobar bronchus and is easily found by bronchoscopy This was the beginning of prope localisation of lung disease. It led to a no nenclatur of lung division based on the position of the broncho-pulmonary segment within each lobe as found by the study of films and bronchograms in rious positions

has vacated is filled by the upper and mid lobes which enlarge by emphysema so that their blood supply is splayed out as against its normal distribution see Fig (7)

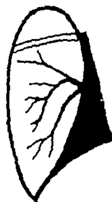


FIG. 7

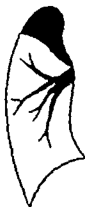


FIG. 8

Fig 8 shows the effects of collapse of the right upper lobe. There is no sign of the blood supply to the collapsed area that to the middle and lower lobes is spread laterally as against the normal distribution

Now and again we come across subjects in whom the normal division of the bronchi has gone wrong Development has ceased at one point of bifurcation and instead of a branch we have an air containing space blown out by air from the bronchus and kept open by surrounding lung structures It may be small or large and is lined by the normal elements of the bronchus It is known as a congenital cyst of the bronchus It can be found in forms which maintain their connection with the original bronchus, but many are closed off from the first or later in life Some contain a serous sterile fluid but most are empty The walls are thin but well enough defined and the bigger ones may give demonstrable pressure on surrounding structures When the connection with the original bronchus is maintained through a valvular like flap these cysts may have a clinical significance but the usual form has no effect on the subject On the film these appear as single or multiple circles sometimes superimposed on each other like heaps of thin curtain rings

We need not know all the details of the finer bronchial subdivisions but we must know the principal branches and their main

respiratory infection the former area will be infected where the patient is lying on his back and the latter where he is lying on his side Further we shall be aided in treatment as we know the exact location of the abscess we know how to position the patient f r postural drainage where to insert the bronchoscope for bronchoscopic drainage or where t operate for surgical drainage

Similarly we have learnt that bronchiectasis is not necessarily a lobar disease We now know that the lingular segment only of the left upper lobe is frequently in olved that the upper segment of a lower lobe may be entirely clear lthough its posterior segments are grossly diseased and that only segmental divisions of the main lobar parenchyma may be invol ed This is of material importance t the patient and to the thoracic surgeon who can now adapt his technique to deal with disease of less extent than one lobe

There are four bronchi of particular interest to the student all are in the right lung, three connected with the main upper lobe bronchus and one with the main lower lobe bronchus

The right upper lobe r epi rterial bronchus has three main divisions the apical the ax llary and the pect ral The apical branch goes upwards outwards and a little backw rds It supplies that part of the uppe zone of the lung which is weak in supporting structure and so is common sit for emphysema and f r the first signs f lobar collapse from bronchial blockag Its air containing cells soon fall n when they re not kept fully supplied by residual l which is thei principal method f maintaining their position They may get stretched and become mphysematous by their lack of upport and when they rupture by uch stretching may cause a t ring f the isceral pleura to produce spontaneous pneumothora This type of pneumothorax is known

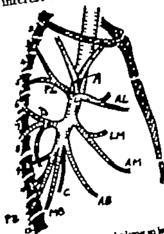


Fig. 11 Bronchi of right lung in lateral view to show relations of branches referred to in text

A, apical AB, anterior basic AL, antero-lateral, ith axillary and pectoral branches AM, anterior middle C, Cardiac; Dorsal (posterior horizontal & dorsal) LM lateral middle MB, middle basic PB, posterior basic PL, postero-lateral (posterior apical, with dorsal branch)

The actual shape and size of the major segments differ but the pattern of each has individual characters so that each lobe can be divided into recognizable areas There are nine of these in the right lung and eight in the left lung The lingular section of the left upper lobe is comparable with the right middle lobe other changes in the pattern of the left lung are due to a combination of two segments in the upper and two in the lower lobe

Abnormal shadows seen on films which approximate to these known areas and bear similar relations to the bony thorax can therefore be identified as disease conditions in definite segments It is always necessary however to supplement this opinion with bronchoscopy One of the great advantages of this method of localization is that a segmental shadow *now focuses attention* on its supplying bronchus The shadow may be due to collapse on consolidation the bronchus will almost certainly be found to be abnormal either blocked or constricted and what is of material importance to the patient, the cause may be something removable

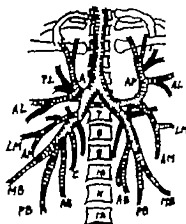


Fig. 10 The anterior view of the bronchial tree, showing the main branches to the lung segments named with them.

A, apical AB, anterior basic AL, antero-lateral AM anterior middle; AP apico-posterior, C, Cardiac; LM, lateral middle MB, middle basic; PB, posterior basic PL, postero-lateral.

The benefits to the diagnostician are very marked Thus through the work of Blair we know that the lesions of pneumonia seldom stick to segmental boundaries they either affect an area less than that of a whole segment or they overflow into adjoining segments so that a truly segmental shadow is against the diagnosis of simple pneumonia It is also against the diagnosis of phthisis except in its bronchial form but truly segmental shadows occur in childhood tuberculosis where *bronchus is constricted by an* enlarged gland

We now realize that lung abscess is frequently found in the upper posterior part of the low lobe and the anterior portion of the upper lobe We see why this can occur from gravity in upper

respiratory infection the former area will be infected where the patient is lying on his back and the latt r where he is lying on his side Further we shall be aided in treatment as we know the exact location of the abscess w know how to position the patient for postural drainage where to insert the bronchoscope for bronchoscopic drainage or where to operate for surgical drainage

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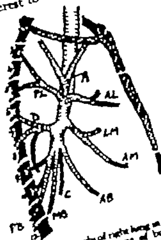


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This type of pneumothorax is known

as simple to distinguish it from pneumothorax of tuberculous origin

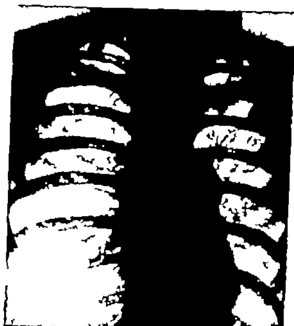


FIG. 12. Simple spontaneous pneumothorax, with emphysematous bullae on the upper border of the collapsed lung

The apical branch is not often affected by pneumonia but is especially important in that it gi es off a dorsal branch supplying a sector of the parenchyma commonly affected in adult tuberculosis When this occurs we see in the supraclavicular area of the lung field small rounded deposits more or less defined in outline and dense in the centre

Reference will be made later to the difficulties in int rpretation of shadows in this area In the meantime it is sufficient to say that it is a fairly safe rule not to read evidence of apical tuberculosis unless one can see quit definite areas of loss of translucency or at least fiv rounded dots lying under internal or external to the shadow of the anterior end of the first rib above the collar bone

The axillary branch comes off th main trunk close to the pectoral branch towards the centre of the upper mid zone of the right lung field and i directed outwards to supply the outer area of the lobe below the clavicular zone



Fig. 13. Apical tuberculosis of the left lung.

When this branch is involved in such a disease as pneumonia we have therefore on the film triangular rea of loss of translucency which has its apex at the entrance of the branch, and its base along the axilla. Like th apical branch it gives off a dorsal bronchus important in adult tuberculosis.

Later in the section on pplied pathology w shall discuss the differential diagnosis of the film shadow of partial pneumonia m of ing the axillary bronchus and f adult pulmonary tuberculosis t the out base of the ight uppe lobe.



Fig. 14. Showing pneumonia of the pectoral and axillary branches of the right upper lobe, and cysts dorsum of the right lower zone.

The pectoral branch bends a l ttle outward and then comes forward in semic rcula fashion the a ching f the ac ompanying branch of the pulmonary rt ry sometimes show up prominently in complet ly normal film as if it wer half cav ty w ll the othe half being f rmed by the mediastinal shadow. If the lung sector of this bronchus is m lv d i pneumonia there is triangula loss of translucency rather like r irror image of the a llary sector this time the base l against the mediastinal

shadow and the apex towards the centre of the upper mid zone of the lung In some cases the shadow is first noted when the apex

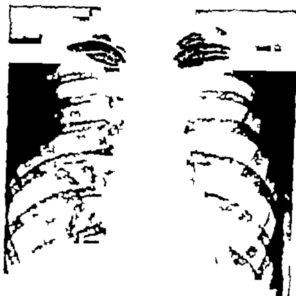


FIG. 15 Pneumonia of the axillary branch of the right upper lobe bronchus.

of the triangle is rounded off this may cause considerable difficulty in diagnosis as against tumour or cyst Serial films usually clinch the diagnosis by the comparatively rapid changes that occur in the partial pneumonia as the disease resolves

The principal branch to remember from the right lower lobe bronchus is the posterior horizontal (dorsal or first dorsal branch). It does exa tly what its name describes It comes off the main stem of the lower lobe bronchus posteriorly prcticlly opposite the origin of the middle lobe bronchus and therefore near the centre of the hilar hadow or the film It goes almost horizontally back wards If therefor it is involved in disease the abnormal shadow is close to the hilum This is why so-called central pneumonia commoner in children than in adults appears to be part of the mediastinum As it hooks backwards over the apex of the lower lobe this branch is a common sit for blockage Its lung segment is often involved in adult phthisis Septic material is easily caught in it to gi e the foundations for an abscess whilk the

accompanying lymphatics round its blood supply are equally easily occluded by phagocytes containing tubercl bacilli

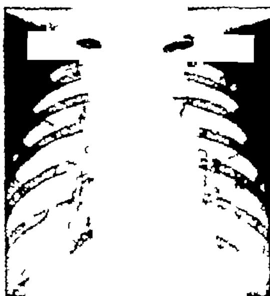


FIG. 16. Pneumonia of the pectoral branch of right upper lobe bronchus.

Its occlusion is a common cause of charact ristic film of adult tuberculosis When a tuberc loma has formed in this area, lose inspection of the postero-anterior film will often reveal a line running downwards and outwards from the hilum and form ng the outer boundary of a triangular loss of translucency The lingula of the lower lobe has shrunk downwards and inward on to the main interlobar fissure There is collapse of the apex of the lower lobe



FIG 17

From the foregoi g outline it will be seen that broncho-pulmonary segments supplied by divisions of the bronch that go backwards are sites of election f adult tubercl we must know particularly the dorsal branches of the apical axillary and pectoral divisions of the epiarterial bronchus and the posterior horizontal branch of the right lower lobe bronchus

They are similarly the earliest sites for evidence of blockage of lymphatics in diseases of dust inhalation such as silicosis The

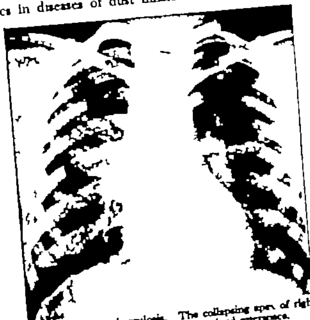


FIG. 18. Pulmonary tuberculosis. The collapsing apex of right lower lobe as tongue-shaped shadow in third interspace.

broncho-pulmonary a eas supplied by these branches are like all such areas conical in shape but on the postero-a terior film are photographed along a line drawn through their narro est point at the entry of the bronchiole

It follows therefore that disease in them throw a rounded or oval shadow because the bronchus and also theref re the segment of lung it supplies lie antero-posteriorly in the chest An ant ro-posterior film (*i e* one with the patient s back to the screen) will show how the diseased area goes backwards The shadow is much bigger

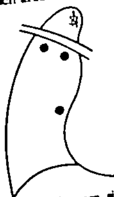


FIG. 19. Common sites of tuberculosis in b n h -pulmonary segments supplied by d lly l d bronchi.

It is important to note that the right middle lobe bronchus comes off the right main bronchus at an angle of almost 90 degrees and that its branches go out almost horizontally towards the periph ry of the lung This makes the sectors of parenchyma supplied by its bronchioles appear as small triangles with their apices inwards and their

bases outwards when they are affected by disease. It also makes them as the main stem difficult to drain and any of blockage

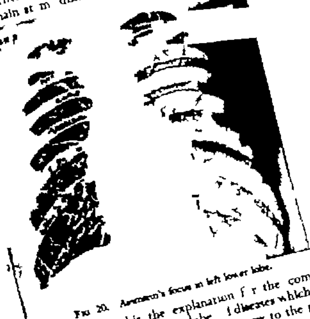


Fig 20. Assmann's focus in left lower lobe.

and we shall find in this the explanation for the comparative frequency of complications in this lobe of diseases which in other lobes of the lung give no such cause for worry to the physician.

We have already noted that all branches of the bronchi carry throughout the lung elastic tissue continuous with that of the trachea. By this elastic tissue they are able to return to their normal position of rest after they have been elongated and opened up in the act of inspiration. It follows from that by such distribution the elastic tissue is anchored to the hilum, so that the lung is always, in filling, the more it is pulled outwards to return to this point of anchorage.

Similarly all branches carry the tracheal structure of cartilage and muscle and have round them sheaths of connective tissue supporting them but moving with them in respiration. These sheaths carry the blood vessels from the bronchial artery and are closely related to the accompanying circulation from the pulmonary artery. Through them run also the lymph channels. The sheaths send out strands which support the lung parenchyma. We shall consider the connective tissue in more detail when dealing with the structure and function of the alveoli.

CHAPTER II

THE ALVEOLI THEIR STRUCTURE AND FUNCTIONS

WHILE the bronchi continue to divide they retain their component cartilage muscle and elastic fibres into the final subdivisions *called the terminal bronchioles* There is much argument among research workers on the details of what exactly happens thereafter until we reach the air cells or alveoli but what matters to the student is that while all the bronchial elements remain up to the terminal bronchiole, the cartilage disappears after this subdivision but strong muscle and elastic fibres continue into the final cylindrical channel the bronchiolus respiratorius This bronchiole with its communicating air-cells forms that minute section of lung called the acinus

The Acinus



FIG. 21 The Acinus.
1 Termination of cartilage.
2 Termination of muscle fibres.
3 Termination of elastic fibres.
A. Terminal bronchiole.
B. Bronchiolus respiratorius.
C. The acinus.

The acinus is so important in the understanding of many diseases of the chest and is the basis of explanation of so many physical and X ray findings that we must pay some particular attention to its details of construction and its relations both anatomical *and physiological*

Its bunches of air cells or alveoli contain the residual air of the lung that is the air that remains afte a maximal expiratory effort has been made to expel what is called supplemental air In other words this residual air can be expelled only by collapse of the cells It is in contact with cubical epithelium, which has replaced *the columnar ciliated epithelium* of the bronchi, and through it is brought into close contact with the finest capillaries of the blood supply for gaseous interchange

Residual air is at atmospheric pressure being the same as that

of inspired ai and therefore the same as that continually pressing on the outside of th thoraci cage In other words, it is one offset to the attempt of outsid air to push in the bony thorax The same ultimate ffect would be got by any other occupying material of the alveoli g pneumonic exudate as long as the pressure is the same This we shall see is an important point in the differential diagnosis of chest disease

The muscle fibres continue along the bronchiolus respiratorius and get steadily stronger until we reach the last passage that can be said t be lumen where we must not they finish by becoming circular only instead f longitudinal and circular This anatomy explains many fundamentals of normal physiological lung movement and of the changes instituted by disease processes By the act of inspiration there is a lengthening of the muscle fibres throughout the whole bronchial tree as far as the acinus thus there is tretching and opening up of every air-containing lumen The muscle is smooth muscl and so maintains movements along the bronchi even when the lung is t rest thus keep ng up th flow of ai to th al eoli with an action imilar to that of the bagpipe

Destruction of muscl in the bronchial w ll is the efore of serious consequence to the acini their residual air is not maintained and they are apt to collapse Muscl spasm in su h disease as asthma has also serious effect the teady spasm induced by the disease even during lung rest is accentuated by the mov ments of respiration, and is communicated to the rcular fibres which lose n vic l ke fashion over the entranc to the acinus The patient has theref re increasing distress by his d fficulty in ma ntaining sufficient residual ir Such an dd tion to his lready established emphysema e plains the xtreme cyanosis often noted in the b onchial asthma of the chroni bronchitic This point will be bett r understood when we consider the effects of emphysema

Elastic tissue continues ll the way to the l eol finishing round each as if it were net bag round tennis ball C nt nuous from the main bronchial division in the mediastinum which may be considered as its point f anchorage it is kept on onstant stretch in ev ry direction throughout the lung fields by the air in the al eoli What this comes t is that the residual ai I each l colus is l k a small distensible bladder which has two physiological effects I rst it keeps up tension on the lastic tissue so that the pull on the anchorage is onstant and therefore the lung is lway attempting to retract towards the h lum

Second it has a marked effect on the strength of the outward pull of the pressure in the pleural cavity which is below that of the atmosphere and is therefore called the sub-atmospheric or negative intrapleural pressure The maintenance of residual air at a definite level is thus a necessity for proper physiological working of the lung both at rest and during the act of inspiration If it is cut off by blockage of its supplying channel or destroyed by the breaking down of its alveolar confining walls we shall find definite changes in the position of the lung and the mediastinum A few examples will h lp us to understand the ultimate effects of certain disease processes by noting the physiological effect of their pathological changes on lung elasticity

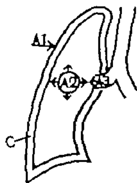


FIG. 22.
A1 Air pressing on the chest wall.
A2. Air in alveolus.
A3. Inspired air in main bronchus.
C. Inter-pleural space.

(*a*) Destruction of elastic tissue along the bronchial wall occurs in bronchiectasis and tuberculosis As some of the tension is thus removed from the point of anchorage of the elastic tissue at the main bronchial division in the mediastinum the lung medial to such a break in continuity tends to contract towards the hilum

(*b*) Destruction of muscle in the bronchial wall is seen in tuberculosis and bronchiectasis air is not maintained in those lobules dependent for supply on the affected branch because the bagpipe action of the smooth muscle has been lost and the end result is the same as in (*a*)

(c) Cutting off residual air which maintains the stretch on the elastic surround of the alveoli happens if anything blocks the supplying lumen—pus blood or a foreign body This happens in adult phthisis Tuberculous material from diseased alveoli enters and later blocks the bronchiole supplying them Empty air cells then collapse those that still contain material organize and all lose their outward pull on the mediastinum

The acini are bound together into lobules by connective tissue septa This connective tissue which runs throughout the lung as a sheath accompanying the bronchial distribution acts as the supporting structure It binds bundles of air-cells together into lobules and acts as a barrier which tries t confine disease to

these small sectors of lung substance This function it can perform because it is rich in blood supply which increases markedly in response to irritation thus giving inflammation as the evidence of resistance Continued inflammation however brings much increase in cells which are later permeated by new capillaries from the swollen blood vessels The result is the formation of fibroblastic cells which obliterate the new capillaries so that nature s attempt at repair of the damage leads to interstitial fibrosis Such repair tissue is fraught with danger to the bronchi and the parenchyma What was formerly a resilient supporting structure is now a fibroid and restricting band which impedes the normal expansion and elongation of the bronchi with inspiration It also interferes with the blood supply of the bronchial artery which runs through it Further by its shrinkage it pulls on the surrounding lobules through thei supporting septa with which it is connected In its advanced stages uch as are seen in bronchiectasis this traction can actually obliterat alveoli by crushing them This combined action of constriction of the bronchi and destruction, and consequent collapse of near by lobules is well demonstrated in the film of early bronchiectasis We can see that the bronchi of the inner bundle of the right lower lobe are crowded together just outside the lower third of the right cardiac border Thei outlines are congested and enlarged so that they are much more prominent than normal and w know there has been destruction of the lobules in thei immediate neighbourhood because w find ther is loss of lung markings in th out low r zone of the lung where compensatory emphysema has result d

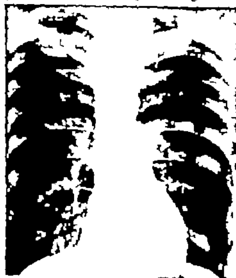


FIG. 21. Early bronchiectasis. Crowding of bronchi in right cardiophrenic angle.

Later we shall learn that increase in connective tissue round the bronchi is one cause of the increase in the normal striations on the film, and that it gives characteristic dry crepitations when we listen by stethoscope

The Supporting Structure of the Lobule

The connecting septa round lobules can be so distended that it breaks down in the disease of emphysema This can be recognized on the film as a loss of normal markings to this point we shall return more fully later in our studies on applied pathology It can also be broken do vn by septic material in its blood vessels when this is carried to them as emboli from an infected focus In this way many small scattered abscesses can appear throughout the connective tissue over large areas of the lung in the disease of pyæmia

Lymphatic Supply

Lymphatics are in abundant supply in the connective tissue *round the lobules Their presence explains the first steps in the* pathogenesis of certain lung diseases The whole system in the *lung is divided into two sets a superficial and a deep* which communicate with each other in the pleura and at the hilum *The superficial set lies in the pleura which we shall consider later* The deep set runs with every subdivision of the bronchi the *pulmonary artery and the pulmonary veins and so it follows that* the connective tissue between the lobules has an exceedingly rich *lymphatic supply This connective tissue upply is finally li ked* with lyn ph spaces that bathe indi idual acini and of this fact we *must make especial note* as it is by this route that tubercle bacilli finally enter the lung parenchyma in adult phthisis The bacilli *circulate and multiply in the lymph spaces* from which they penetrate the walls of the air-cells

All lymphoid tissue in the body is meant to be a protecting facto both at the point of any disease process where it acts as an *absorbent agent* and also in its canal ystem whereby actual body irritants and their products may be carried to the glands *In other word* the glands are the drains the lymph channels are the gutters We see these lymph channels act as a conveyor *system in silicosis* they transport dust-containing cells from the alveoli to the lymph spaces round the acini from there to the

connective tissue lymphatics and thence by peri-vascular and peribronchial channels to the lymph glands Lymphoid tissue carries out the same function in childhood tuberculosis draining the primary focus to the glands in the hilum But in adult tuberculosis of the proliferative type w see that it is the lymph spaces between the alveoli that start the first reaction towards the production of the acinar lesion not only d the lymph spaces fail to perform thei proper function in this case but they actually break down to become culture ground for the in ading bacilli All lung lymphoid tissue increases with age It is the breeding ground f r that widespread form of tubercl known as senile phthisis But it seems to do its utmost to reassert its protective powers The patient is so littl upset constitutionally that he may be a dangerous but unsuspected carrier of the disease and is often treated for what he appears to be a chronic bronchitic

The Blood Supply

The al eoli re ery rich in blood supply the final capillaries of the pulmonary art ry being so numerous as t be component of their w lls while their connecting and binding epta of onnective tissue has an almost equally abundant supply f nutritive essels derived from the bronchial artery The distribution of these arteries explains many points of difficulty in chest diseases and helps us to und rstand many of the markings on normal and abnormal diagrams

The function of th pulmonary artery is oxygenation of the blood stream It originates from the right entricle and under the rch of the aorta di ides int its right and left branches At the hilum the right branch l es in front of the right main bronchus the left branch just bo the left main bronchus It follow there f re that the main pulmonary artery and ts branches enter in great part into the composition f the hilar hadow on th film In the lung each artery f llows the bronch lying behind and lightly lateral to them in all their sub-divisions They are the guide as w noted n the last hapter to the bronchial distribution. W must remember howev r that while close to the h lum the art ry and the bronchus are practicall the same ize the artery lessens in size ery fast as compared with the bronchus and gi es ff many more branches within the same distanc this is why when looking at an abnormal film w can t ll whethe the radial lones of translucency we see in streak a blood essels or bronchi

Within a distance of an inch towards the middle of the lung fields a bronchus might be seen as continuous parallel lines with no branches while an artery will not so appear By the time the pulmonary arterial sub-division has reached an acinus it has only about one fifth of the diameter of the supplying bronchiole to the alveolus

We must remember however that the arteries can be materially changed in size by various diseases Thus in congestive failure they may be considerably swollen -they then give increased soft markings generalized throughout the lung fields and lessening towards the peripheries of the film The opposite effect can be noted in conditions associated with extreme anaemia where their thin flaccid tubes are indistinguishable from the normal striations of connective tissue These changes will of course be accompanied by relative changes in the shape and size of the cardiac shadow *and be apparent in both lung fields*

The function of the bronchial artery is to bring necessary nutrition to the framework of the lung That for the right lung comes usually from the thoracic aorta but occasionally from the first or third intercostal artery the left supply has usually two origins from the thoracic aorta. It is important to remember this difference of origin of the pulmonary and bronchial supplies the latter as direct from the main arterial vessel has the *higher* pressure so a wound or other cause of a break in its wall can prove much more rapidly fatal than the involvement of an arteriole from the pulmonary artery

As soon as the bronchial arteries enter the lung they enter the connective tissue layer round the bronchi two or three subdivisions accompanying each bronchial branching This is why any condition increasing blood supply has the *possibilities of* marked effects on the bronchial walls why such conditions of *long-standing inflammation as chronic bronchitis* and bronchiectasis can cause the havoc of fibrosis round the bronchi affected in the disease Here we have the explanation for the film of chronic bronchitis The normal supply from the bronchial artery is very rich When this supply is swollen by chronic inflammation, and brings the consequent deposition of fibroblasts we get the evidence *on the* film in the appearance of parallel lines in the affected area outlining the larger *bronchi in the lower zones of the lung* fields We can find the same effect in other parts of the lung in other diseases—*g* in the upper zones in chronic pulmonary tuberculosis

Such rich blood supply is the reason why quite severe hæmorrhage can occur by even small abrasions of the bronchial wall by irritant material inspired into the smallest divisions of the bronchial tree; and why the breaking down of the bronchial wall in bronchiectasis can produc the biggest of ll hæmorrhages in lung diseases

CHAPTER III

THE PLEURA AND THE DIAPHRAGM

THE pleura is in two layers connecting at the hilum The first or visceral layer is closely applied to the lung It reflects at the hilum to form the second or parietal layer which lines the inner surfaces of the structures internal to the thoracic cage, and the diaphragm The parietal layer is sensitive Pain and severe shock can occur when a needle is pushed through it in the induction of artificial pneumothorax, and the pull of adhesions on its inflamed surface may well be the reason for the acute stabbing pain with each respiration in lobar pneumonia

The two layers have between them a potential space They do not adhere in health but move easily on each other by virtue of a thin lining of serous fluid in which the visceral layer can slide on the parietal In the space there is a pressure which is negative in that it is below that of the atmosphere a needle inserted in this space and connected with a water manometer shows that the pressure varies on an average between —10 cm of water pressure with inspiration and — 5 cm with expiration It th refore exerts a constant outward pull on the elastic tissue of the lung, adding to that of the residual air in the alveoli each inspiration adds to the pull *thus lengthening and opening up the bronchi to receive the* inhaled air

It follows that any pathological alteration of the negative intrapleural pressure has a marked effect on the elastic tissue of the lung If it be reduced to the level of atmospheric pressure by ai in pneumothorax or fluid in pleural effusion the lung must tend to recede to the anchorage of its elastic tissue at the hilum retracting to size less than that of complete expiration *The* stretch on the elastic tissue of the lung is now maintained only by th residual alveolar air Raising of the pressure bo e that of the atmosphere and therefore above that of residual ai will bring pressure collapse of the alveoli and consequent still further retraction of the lung for now the pull of the alveoli on the bronchial elastic tissue is being lessened As we shall see later when discussing the mediastinum these are the ruling factors in the displacement of mediastinal structures in pleurisy with effusion

In going round the lung as described the visceral pleura dips between the lobes lining the under surface of the one above and the upper surface of that below it. One such lining may appear on a normal postero-anterior film of the chest. It is the lesser fissure between the upper and middle lobes of the right lung, and it shows as a thin hair lik line running out almost horizontally from the hilum in the third o fourth rib-interspace. Sometimes it casts a double line as the X rays have cut across it tangentially. Its position is easily recognized when it is forced apart by fluid or its rich lymphatic supply is inflamed or thickened n such diseases as pneumonia lung abscess and tuberculosis. If it is seen on the postero-anterior film its location and direction hould be noted carefully as this may giv an indication of disease process that can be clarified only by further film examination—e g lateral film. Thus it is displaced upwards and towards the infra-clavicula zone in collapse of the right upper lobe.

Other fissures do not show on the normal postero-anterior film and it may tak lateral or lordotic films to show their involvement in pathological conditions.

Some tudents find d fficulty in the interpretation of abnormal films because they forget that the interlobar fissur as they visualize them are but the points of r flection of the pleura which in completely ...
...ing any lobe must necessarily cover by the visceral lay...

The shadow cast by normal lung being but the uperimposed shadows of it ... rlying structures from front to back on the postero-anterior film it follow that density of hadow in dev loped lobar pneumonia aries by depth of tissue and not by intensity of disease which is equal in the bod and the lingula of the ffected lobe.

It ... cannot expect the shadow of right upper lobe pneumonia to be as dense in its lower limit as in it upper because the lobe thins out considerably as it goes downwards. w recognize middle lobe pneumonia because it has a dense hadow in the right mid zone which lessens st adily t w rds the base so that th ough its low lim ts w an see the right diaphragm.



FIG. 4. Right upper lobe pneumonia.

This explains too why collapsing lobes cast characteristic shadows why the lesser interlobar fissure throws a heavy density upwards and outwards as the lingula recedes on to it in right upper lobe collapse Likewise we realize that when a right lower lobe collapses the lingula has already shrunk downwards a long way before we see on the film the upper limit of the lobe defined by a line running downwards and outwards towards the costo-phrenic angle from a point at the upper edge of the right auricle on the right side of the cardiac shadow (see Fig 26) The position of these fissures in the lateral film is discussed in Chapter V



Fig 25 Pneumonia of the right middle lobe, and of the left lower lobe.

With such changes in shape of lobes we shall learn to correlate changes in the normal striations that appear on the film by shadows of vascular supply and supporting structure Thus we have already noted that in right upper lobe collapse we can no longer see the three normal branchings of the pulmonary artery which supply it, while in the rest of the lung field we shall find spread out much more horizontally than normal the blood vessels of the middle and lower lobes which have been blown out by complementary emphysema to occupy the space vacated by the shrunk upper lobe In this emphysematous area we shall at the same time find much increased translucency as against the normal because the supporting structure of the vesicles is considerably stretched We therefore make our diagnosis by noting two opposite processes: increased density and increased translucency; one with loss of blood supply the other with spread of blood supply and of supporting structure In other words where we suspect a change in one type of lung striation we must look for a corresponding change in the other and if we fail to find one in the presence of another on a postero-anterior film we must make other investigations such as the taking of lateral films or the injection of lipiodol Thus in complete collapse of the left lower lobe we may see on the postero-anterior film only the gross emphysema of the upper lobe filling the

hemi thorax we may not be seeing the collapsed lobe because it is completely hidden by the cardiac shadow films taken in the postero-anterior and lateral positions before and fter the injection of l piodol will aid in the diagnosis

The Diaphragm

The diaphragm is the most important muscle in respiration and therefore next to the heart the most important muscl fo the maintenance of life It consist of thin, mov ble partition lying between the thorax and the abdominal contents It is convex upwards its right half being slightly higher than ts left and taking a position just below the nipple at respirat ry rest There is a central tendinous very trong portion and three fleshy portions which me from the ribs th sternum and the spin and are therefore named costal t rnal and vert bral Of these we must note especially th costal; its origins are from the inner surfaces of the lower six costal cartilages on each sid When th diaphragm is depressed they may be seen on a postero-anterior film in thel diaphragmatic insertions as depressions between convexities thus f rming series of w es and such a picture while commoner in emphysema does not nece sarily connot ny disease process They ar sometimes wrongly interpreted as evidence of adhesions of old pleurisy If there is really adhesion of the diaphragm there will be no clear outline but merging of lung shadows into the deeper density f the muscle

These normal alterations in the dome may also be confused with the ery common finding of tent lik shadow that its on the diaphragm about its middl th rd This is the end result of pleural reaction in the main fissure

The central tendinous portion may appear as a hump on the middle of the diaphragm it is without linical ignificance but may require investigation against the comparati ly rare pleural growth that can develop at this point



FIG. 26. The costal insertion of he diaphragm.

By the contraction of its fleshy portions through the action of the phrenic nerv the diaphragm becomes less convex and so increases the ertical diameter of the thorax in inspiration In ordinary breathing this range of movement is bout three-quarters

This explains too why collapsing lobes cast characteristic shadows why the lesser interlobar fissure throws a heavy density upwards and outwards as the lingula recedes on to it in right upper lobe collapse Likewise we realize that when a right lower lobe collapses the lingula has already shrunk downwards a long way before we see on the film the upper limit of the lobe defined by a line running downwards and outwards towards the costo-phrenic angle from a point at the upper edge of the right auricle on the right side of the cardiac shadow (see Fig 16) The position of these fissures in the lateral film is discussed in Chapter V

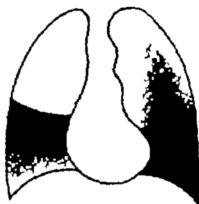


Fig. 25. Pneumonia of the right middle lobe and of the left lower lobe.

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FIG. 24 The costal insertion of the diaphragm.

By the contraction of ts fleshy portions through the action of the phrenic nerve the diaphragm becomes less convex and so increases the ertical diameter of the thorax in inspiration In ordinary breathing this range of movement is bout three-quarters

of an inch and in every examination of the chest it should be looked for by percussion of the lung bases posteriorly and in the axillae since deep inspiration should move the diaphragm downwards by at least a finger's breadth as shown by the higher pitch of descending air-containing lung

It follows that interference with the phrenic nerve at any point throughout its course over the cupola and past the hilum to the insertion of its fibres will cause rise of the diaphragm Evidence of this is common nowadays in the films of patients in whom the nerve has been crushed or evulsed as a treatment for pulmonary tuberculosis Such films demonstrate that the diaphragm as it moves downwards in inspiration exerts a distinct pull on the apex of the lung, for when it is immobilized an apical tuberculous lesion shrinks and gets the rest it requires to aid healing

Adhesion between its pleura lining and the visceral pleura of the lobes in contact with it will fix the diaphragm while emphysema will depress and flatten it Contraction of the lung by loss of elasticity will cause it to rise *e g* in collapse of the right middle lobe The recognition of such changes in its position and movement by physical and film examination can therefore be of very distinct help in the diagnosis of underlying lung disease

CHAPTER IV

LUNG AND MEDIASTINAL MOVEMENT IN HEALTH AND DISEASE

THE mediastinum is a space running antero-posteriorly in the mid line of the thorax between the sternum and the spine It shows on the postero-anterior film as the shadow of parts of its contents so that we can identify the trache to a point just above its bifurcation the outline of the cardiac hadow the aortic notch, and the structures that mak up each hilum As w ha already noted all these mediastinal tructures re embedded n areolar tissue which covers and binds them together but yet is elastic in composition, so that it can mov with syst le and diast le of the heart and the great blood essels

W have already noted too that it is connected by the lay r of pretracheal fascia with the investing fascia of the structures in the neck and that thereby its displacement to one other sid of its central position is reflected to an increased tension of the tendinous portion of the st monastoid muscle on the same side

Again w saw that the mediastinum has a w ak point on which t wings as on hinge and that the trache can mov laterally both abov and below this hinge for example that t can be definitely kinked by disease localized in the parenchyma below the collar bone The heart is displaced much less read ly A much heavier tructure t has its greatest w eight in the lower medi-astinum so that t is moved laterally only by disease cond tions n the lower half of the thorax or throughout one whole sid of the thorax Thus the apex beat is seldom bnormal in position in tuberculosis of one upper lobe but it can be mo ed quite markedly t the opposite side in the presence of pleural effusion and to the same side in post-pneumonic fibrosis this being disease which commonly affects the whole of one lung field

An mportant point to not is that the heart w ll wing round its long axis much more readily than t will move laterally This explains the anomalous outline t presents in th normal abnormality of scoliosis which has its usual concav ty to the left In lying into this hollow the heart swings on its long axis ts right border

receding and its left border coming forward so bringing the pulmonary conus on the left side into undue prominence and

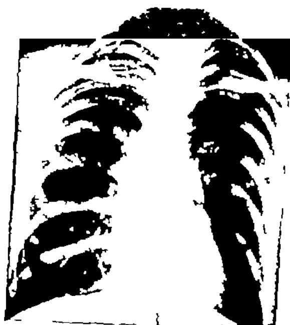


FIG. 27 Marked scoliosis with convexity to the right.

uncovering the right hilar region On the postero-anterior film there is therefore a filling in of the normal triangle of translucency that has its apex between the aortic notch and the normal prominence of the conus giving an outline that simulates that of mitral stenosis on the left side while the shadows of the right hilum and of the branches of the pulmonary artery to the right lower lobe stand out prominently Such abnormalities might be read as evidence of disease if we did not note the accompanying widening of the right lower rib-interspaces and the compensating fall-in of the left lower rib-interspaces We can see too why it is by this tendency to swing on its long axis that the position of the heart at respiratory rest may mislead us in such a disease as collapse of the right lower lobe As it shrinks from such a cause as bronchial carcinoma the lobe goes downwards inwards and backwards and causes the heart to swing on its long axis so that the right lower

border recedes towards the spine the left lower border comes forward Lat r the whole heart goes backwards If we have to rely on physical signs for our diagnosis w shall often find more help towards the opinion that the mediastinum is affected by noting the tense right sterno-mastoid than by concluding anything from the position of the apex beat If w can watch the patient on the screen however we shall see the heart move laterally and bodily to the right in its lower half with each inspiration and come back to its former position with expiration This is because the intra pleural pressure becomes more negati e at each inspiration and drags the whole mediastinum towards the periphery

A little later we shall see why the heart goes backwards in collapse of the right or left lower lobe

Experience show that mobility of mediastinal structures aries considerably in different individuals The pressure in the pleural cavity in two subjects may be the same as judged by the same manometer in conditions as nearly equal as we can mak them yet the amount of displacement from normal n disease of eemingly equal extent may be entirely different In one small pneumo-thorax can aus great bulging of the ant rior mediastinum in another it will hardly move this area This means that the mediastinum is never a fixed and invariable ent ty w cannot by working backwards gi an infallible estimat of the amount f lung disease by basing it on the amount of mediastinal shift and all we can say is that some damage has taken place On the other hand w must realize that diseas of the mediastinal pleura can combat peripheral changes of pressure Thus we may have definit stiffening effect thickening of the mediastinal get much more falling in of ribs and raising of the diaphragm than w would expect from the amount of damage evident in the parenchyma

The mediastinum must b) be envisaged as one entity not as something in two halves one for ach hemi-thorax It is one central lastic tructure both maintaining and being maintained by one intra-thoracic pressure If there is a fall of pressure on one side of the thorax there is an immediate utomati rise on the other Fibrosis on the right implies emphysema on the left Now w can understand why the he rt goes backwards in right lower lobe collapse The pressure in the right lower mediastinum is lessened It cannot put up normal resistance to the pressure in the left low mediastinum w th the result that the left lower

lobe moves across the front of the heart and pushes it backwards.

We are now in a position to sum up our considerations of applied anatomy to discuss the action of the various lung and intra pleural forces on the mediastinum in health and see how they react on each other and on this central elastic structure in disease

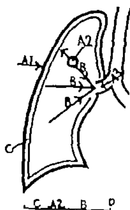


FIG 28 The forces acting on the mediastinum.
A_1 Air pressure on chest wall.
A_2 Air in alveolus.
A_3. Air inspired into bronchus.
B. Elastic tissue
C. Intra-pleural pressure.
D Mediastinum.

In the accompanying diagram, D represents the mediastinum A represents atmospheric pressure which acts in three ways —

(1) As A_1 pressing in on the chest wall

(2) As A_2 the pressure of residual air in the alveoli we have seen that this is maintained by the bag-pipe action of the smooth muscles of the bronchi A_2 acts in every direction throughout the lung fields pressing against the periphery the diaphragm and the mediastinum We ha e seen it demonstrate its force against the mediastinum in collapse of the right lower lobe It also presses on surrounding alveoli so if it is lost in any area the surrounding alveoli immediately distend, giving localized emphysema

(3) As A_3 the air entering by the main bronchus

B represents th elastic tissue of the lung, which runs with ev ry bronchial division from the hilum to th alveoli The more a piece of elastic is stretched the more it tries to contract to its fixed point As lung elastic tissue is on constant stretch by the action of the residual air in the alveoli (A_2) it is always attempting to pull th lung inwards to its anchorage at th hilum, where it is continuous with the elastic tissue of the trachea Therefore in proportion with the destruction of the lastic tissue th lung will always contract towards the hilum

C represents the sub-atmospheric negative pressure in the pleural cavity This exerts steady outward pull on the lung, and is increased with each inspiration

Again we must remind ourselves that all these forces are acting in exactly the opposite direction against the mediastinum on the

opposite side A_1 on the right is pushing to the left A_2 on the left is pushing to the right Thus the central balanced position of the mediastinal structures

Let us now consider examples of disease conditions affecting each of these factors and see how they react on the mediastinum

A is constant factor in all ordinary conditions of life in health and disease

A may lter by such conditions as —

( ) blockage of the bronchus through arcinoma or septic r caseated mat rial

(2) destruction of the alveoli as in tuberculosis of adult type

There is therefore loss of residual air A_1 has less resistance offered to it so the hemi thorax tends to shrink and the ribs tend to fall in There is less resistance to the diaphragm so it will rise higher t rest and with complet expi ation There is less resistance to neighbouring lveoli and so they show emphy sema There is less resistance to the mediastinum so the force on the opposite sid w ll push the mediastinum to the diseased side Finally there is less pull on the elastic tissue anchored to the trachea through the main bronchus and so the lung will retract t the hilum and this means that C is now much less opposed and drags the whole lung outwards W saw n extreme example of this action f C in the detailed discussions f what happens in collapse of the right lower lobe We noted that the lower half of the mediastinum ncluding the lowe half of the heart, wings out to the right with each inspiration, and comes back to more entral position with each expiration

We can represent this in a imple line dr wing (see Fig 28) remembering that ll processes re acting equally in the r verse direction n the opposit hemi-thorax

A is destroyed B therefo shrinks towards D and C now unopposed pulls B and D outwards The mediastinum therefo drifts to the side of the atelectasis, the amount depending on how much of A_3 is destroyed We see now why t is that we get pos tive sternomastoid ign with loss f residual ai It is not due directly to the collapsed tissue but to such lung tissue as remains unaffected and is dragged over bod ly by the intra-pleural pressure Its drag is communicated to the trachea.

If we have followed these arguments w can see why such diseases as unilat ral tuberculosis o bronchiectasis must displac the mediastinum to the side of the lesion

We have here too an explanation of cavity formation There is marked localized loss of elastic tissu by organization and collapse of lobules and therefore loss of the pressure of their residual air Immediately surrounding lobules are unopposed in their own inherent pressure and blow out to become emphysematous and so their weakened walls are dragged in every direction towards the periphery both by those external to them and by the intra pleural pressure Apart altogether from any extension of the actual disease process there is physical force behind the production of increasing cavitation

It is possible to follow now why diseases which replace residual air by material which keeps the alveolar walls distended cause no displacement of the mediastinum unless they overfill the vesicles and so pull the mediastinum a little towards them Pneumonia fills the alveoli with exudate they cannot move but they keep the elastic tissue of the lung on stretch as full inspiration does. Therefore uncomplicated lobar pneumonia does not gi a sternomastoid sign nor does an uncomplicated abscess of lung which as we shall discuss later is merely a filling of lobules by inspired septi material Tuberculosis on the other hand will do so it destroys th alveoli Collapse of the lung does it takes away residual air We ha e therefore here very distinct aid towards diff rential diagnosis

Let us now consider disease conditions affecting C the sub-atmospheric negative intra-pleural pressure If we ha e a case of adhesi e pleurisy C disappears A now attempts to fill the space vacated by C and drags B and therefore D with it we get a positive ternomastoid sign on th same side as the disease

If fluid or air ent rs the pl ural spac to remov the negati e pressure and bring it to zero A_2 alone maintains stretch on B the *lung retracts to th hilum and is not moved outwards by* inspiration But the force in the opposite pleural ca ity is corres pondingly greater and so we have a drag to this opposite side on which the sternomastoid sign will be positive In other w rds it does not require actual push from the right in a right-sided pleural effusion, to produce mediastinal displacement to the l ft

If the intra-pleural pressure rises above tmospheric by in creasing air or fluid it will be greater than A_2 The lung is now pushed in by pressure collapse The outward pull of A_2 is lost to B which retracts still more towards D Thus fluid in the right pleural cavity pushes on the mediastinum and t the same time

th pull from th l ft id is till more strengthened nd we have now very ma ked left t momastoid tension

Much will depend now the condition of B If there is much fibrosis of lung opposing th lasti ty of B th lung will not retract to th mediast num D but only mo e towards D if it is pushed o bodily Thus a pneumothora wh h complicates established pulmonary tuberculosis can gi e much more displacement f the mediastinum than imple non tuberculous pneumothorax which is due to ruptur of bulla The lung without disease collapses qu t readily

If w f llow this explanatio f lung and mediastinal movement we shall be in a better position to understand th physical and X ray igns of those commone chest diseases whose pathological hanges are consid red i Section IV

CHAPTER V

THE NORMAL CHEST FILM

A FULL understanding of what constitutes a normal film of the chest is a necessity for the student who hopes to interpret the abnormal. The following description relates only to the points on a postero-anterior and a lateral film which concern the average practising physician. It does not go into all the finer detail which concerns the specialist chest physician, the cardiologist or the pure radiologist, which detail the student may find in specialized text books. It tries to set forth only those points which the physician can correlate with his physical signs and their causative pathology, so that he can go beyond the merely objective reading of abnormal lung shadows.

Some preliminary observations on prime principles must be set out. Thus we have already noted that a film is merely a composite photograph of superimposed shadows of various structures through which the rays pass. With shadows of disease at any one depth we shall therefore see the shadows of normal structures in front of or behind them. An early adult tuberculous focus is completely homogeneous to tomograph examination and much more defined on an antero-posterior than on a postero-anterior film. On this last film it may appear to be striated because, being a posterior lesion, it is shown with much normal lung in front of it, and such normal lung will now throw its striations in greater comparative relief against the background of the disease focus. In the same way we shall see varying shadows of a bronchopulmonary segment in the lung fields. If it be supplied by a laterally directed bronchiole this cone of tissue will appear on the postero-anterior film as a triangle with its apex at the point of entry of the bronchiole; if it be supplied by a bronchiole that goes dorsally, it will appear circular or oval as photographed along a line between its apex and the centre of its circular base.

A good film is the result of good positioning, proper exposure as judged by muscular development of the subject, and good processing. Although the result is by no means a stereoscopic picture, there is no doubt in the mind of the writer that viewing such a film first at close quarters, and then at a distance of some

5 feet will tell the observer much of the composition of bnormal shadows if not of their depth in the lung fields

Of late years medical literature has shown much less ariation in the objectiv and interpretative readings of chest disease because certain criteria of a good postero-anterio film have been more or less generally accepted These are that it may show the lesser fissure of the right lung in the third o fourth interspace that it should show the superior vena cava with dens ty slightly less than that of the heart, and that t must show the shadows of the branches of the pulmonary arteries radiating from the hila throughout some three-quarters of the lung fields

It must be borne in mind that the hila shad w are themselves predominantly ascular As the pulmonary rteries and eins give the main part of the rather indefin t semi-circular opacity on ach side of the cardiac shadow it follow that this opacity will be increased by all conditions characterised by hyperaemia Seeing the left one is already denser by the prominence of the conus in the normal abnormality of scoliosis such a condition as broncho-pneumonia in scoliotic subject will produce ery heavy left hilar density both during the acute tage of the disease and for long after linical recovery

The Normal Postero-anterior Film

Th first lesson the student must learn is t read ev ry film normal and abnormal on definit pre-arranged plan If this is not done consistently even the expert observer may find his ey has been arrested by some striking area of density or contrast or by one interesting detail in on area of the lung fields and that he has missed an abnormality f lesser extent or contrast that is nevertheless of the first mportance in diagnosis For example a filling in of one costo-phrenic angle is dependent f r the interpretation of its real ignificance on the presence o absence of a lung focus We hav already noted how a grave mistake may be made in the interpretation of th film f a simpl scoliosis if we do not connect the variations in the heart outline and the right hilar shadows with the changes in size of the lower rib int r spaces

A simple plan is to ead every postero-anterior film under the following seven headings —

( ) The thoracic cage
(2) The trachea; hape and position

(3) The heart size shape and position
(4) The diaphragm position and contour
(5) The upper zones of the lung fields
(6) The middle zones
(7) The lower zones comparing the right with the left in each case

The Thoracic Cage

The first thing the student of chest films should learn is to recognize the shadows of the bony thorax and those of muscle fascia and skin that can appear on a normal film

The ribs are seen more clearly or less clearly than normal in certain pleural and lung diseases Tuberculous pleural effusion displaces the lung shadows inwards through the even light loss of translucency cast by the fluid in the periphery the ribs show up more clearly than normal They are still more prominent in the film of pneumothorax In empyema and pneumonia they are less prominent owing to the dense shadows thrown by these diseases

The anterior ends of the ribs often continue into the shadow of calcification of cartilage which is more dense and less even, so



FIG. 29 Fusion of anterior ends of first and second right ribs.



FIG. 30 On the right an under-developed first rib. On the left ball- and socket junction of first and second ribs.

that t can mulat the shadow cast by pulmonary disease especially tuberculosis Usually the ondition is seen n more than one rib lthough it is ommo est in the first rib E en then it is lmost lways bilateral and t is a fairly saf ald to remembe that lung lesions re rarely milar i position and characteristics on both ides of th hest

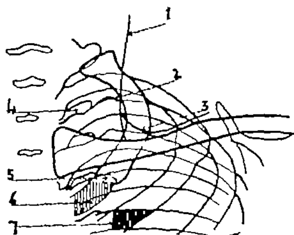
Fusion a d bifurcation f the anterio ends of ribs is ommon Special films may be necessary t discriminat between the hadows nd those of underly ng lung disease Th commonest f all these n rmal abnormal ties is fusion of the anterio end f the first and se ond ribs

Points of int rsection and overlap f rib-shadow an use considerable d fficulty This is especially so at the apices where the shadow of the la icle f the ternomastoid muscle and f skin and fascial f lds complicat



FIG. 31 T show how tuberculous foci may be hidden by b shadows

the picture Fig 32 illustrates how these shadows may be recognized That cast by the sterno-mastoid muscle may need



F G. 32. Drawing to show normal shadows at the apex which may cause difficulty in reading films. 1 = Sternomastoid muscle. 2 = Costal tubercle of third rib. 3 = Shadow due to skin and fascia. 4 = Transverse process of ertebra. 5 = Tip of first rib. 6 = Calcification of first costal cartilage. 7 = Area of density due to superimposition of the shadows of two ribs crossing one another Note also the expansion shown on the course of the second rib as it crosses the clavicle.

careful scrutiny as it lies in the area involved in tuberculosis arising in and around the posterior apical branch of the upper lobe bronchus

Bad positioning causes the shadows of the scapulae to obscure the outer parts of the lung fields but these should be recognized as readily as the rounded shadows due to the breasts in the lower zones in females The shado s of the pectoral muscles and their blood supply may give quite pronounced striations and loss of translucency in the infra-clavicular and mid zones of the lung fields especially on the right side but generally in this case the lower edge of the pectoralis can be seen as a sharp line running downwards and inwards

The Trachea

Th trachea should be seen as a band in front f the vertebrae slightly inclined to th right and with no clearly defined walls. It may be a littl to one side in scoliosis and is generally seen as far as its bifurcation at the level of the fifth dorsal ertebra.

The Heart

An understanding of the normal cardiac outline is a necessity inc it alteration accompanies and can aid in the diagnosis of diseases which are primarily pulmonary in origin The following description does not give deta is required for th tudy of primary cardiac disease

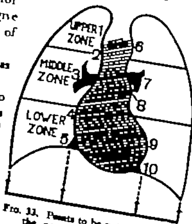


FIG. 33. Points to be noted in reading the postero-anterior film of the chest.

The cardiac outline show as an opaque pear-shaped shadow The transverse diameter is of no pathological significance unless the heart be of the longitudinal type It cannot help where it is of the squat transverse type In this latter cas the outline of the left lower bord is usually indefinit as the heart is pushed upwards by the diaphragm Th tudent should learn to recognize that the hadow of such heart lessens steadily from its mid point outwards and not that under its indefin te inferior border he can till see the density of the diaphragm all the w y t the cardiophrenic angle H will then hav no fear of confusing it with the characterist shadow of lower lobe collaps (compare Figs 86 and 87) Here there will be dense los of translucency filling the cardiophrenic angle the opacity is equal to that of the mid-point of the cardiac shadow with which it is continuous Its outer border is sharply defined The diaphragm does not show as definite linea outline below it, while the costo-phrenl angle is abnormally f ee from trations because it is filled by the emphysematous uppe lobe

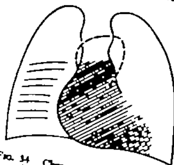


FIG. 34 Chronic bronchitis and emphysema showing squat heart, unfolded aorta, splaying of the ribs and flattening of the diaphragm

Two measurements are taken in assessment of the size of the longitudinal heart —

(1) The transverse diameter of the heart that is the distance between two parallel perpendiculars drawn downwards from the outermost parts of its shadow

(2) The internal diameter of the chest that is the distance between the outermost points of the thorax along a line drawn horizontally just over the right diaphragm

For normality (1) should be just under half (2)

Let us now follow the cardiac outline starting at the right upper border and working downwards and following the same order on the left side

The right upper border is defined by the superior vena cava (1). This is superimposed on the shadow of the ascending aorta (2). It has a light loss of translucency similar to that of the inferior vena cava that is it is slightly less dense than the main cardiac muscle shadow Below it we find the T shaped shadow of the right branch of the pulmonary artery (3) entering into the indistinct semi-circle of the hilum Next comes the border of the right auricle (4) leading to the cardio-phrenic angle This should be easily recognized but not clear-cut with a pronounced edge In its inner angle we can usually see the downward and outward sweep of the inferior vena cava (5)

At the upper left border we find the aortic knuckle (6), and below it the elbow shadow of the left pulmonary artery (7) then the conus (8) and finally the outer border of the left ventricle (9), leading to the cardio-phrenic angle In the angle we may see a slight down vard and outward loss of translucency comparable to thrt of the inferior vena cava this is the pleuro-pericardial pad of fat

In its widest part the normal longitudinal heart is roughly one-third of its width to the right and two-thirds to the left of the mid line of the spine This is why the blood supply to the right lower lobe is so much more evident than that to the left lower lobe and why this normal difference is so exaggerated in the normal abnormality of the usual scoliosis vith its conve ity to the right.

The absol tely normal film has a fairly clear triangl of translucency with its apex inwards above the conus This is the area which is filled in by a line gi ing apparent continuity between th pulmonary artery and th uppe outer edge of the left ventricle when the conus swings forwards in scoliosis

Normal glands do not appear on films indeed ly the tracheo-bronchial group and th broncho-pulmonary group f ry small glands are uncovered by normal mediastinal cont nts It is unwise t diagnose enlarged glands unless they re undo btedly ry prominent They can be demonstrated only f they are so placed that they show against lung tissue which they cov and this means that they must ppear on the post ro-anterior film round th hilum o just und r the right upper lobe bronchus between the aortic knuckle and the uppe borde of the left entricle

The Diaphragm

The diaphragm appears as two clear cut dome-shaped shadow one on eithe ide of th cardia shadow acting as th low r limits to th basal translucency f the lung-fields The right which rises to the ixth interspace is l ttle higher than the left usually to the extent of one int rspace Th blood vessel hadows may be seen to co tinue below it, but neithe in health no disease unless they are injected w th lipiodol do w see the bronchi in this position



Fig. 35. Showing azygos lobe, blood vessels and bronchi end-on, tenting of the diaphragm and obliteration of the costo-phrenic angle.

Th are two common findings that must not be read as evidence of abnormality of linical import The first is so-called tenting of the diaphragm A small tri-angle of short base tting on the diaphragm, and drawn into rathe longated pex is seen towards the inne id of the mid point of one or other half of the muscl This is light pleural reaction in the main fissure and is common finding with broncho-pneu monia even when by the history this condition has been of so low-grad that t has not upset th patient suffi ently t mak him seek advice It remains fo long time en permanently It must not be read as an adhesion as evidence of lung disease unless defin t pulmonary focus is seen when t may be d t contraction of the fibres f the phreni nerv which has been involved disease process in some part of its course ver th cupola and past the hilum Such peaking is nev then t the position of th main fissure and its commonest cause is apical fibrosis usually tuberculous in origin

The second is a filling in of the costo-phrenic angle on one or other side by an opaque shadow This is again a common sequence of a previous pneumonia or broncho-pneumonia the exudate on the visceral layer of the pleura has organized It is not evidence of present lung disease and of no significance unless a lung focus is seen with it, in which case it is usually evidence of a secondary pleural reaction to a tuberculous deposit

The Zones of the Lung Fields

The zones of the lung fields are defined in accordance with the diagram in Figure 33

The upper zones are the areas lying between the upper apices and a line drawn horizontally across the anterior lower ends of the second ribs

The middle zones lie between this line and one drawn similarly across the anterior lower ends of the fourth ribs

The lower zones lie between this second horizontal line and the lung bases

These zones are used to designate the position of any abnormality

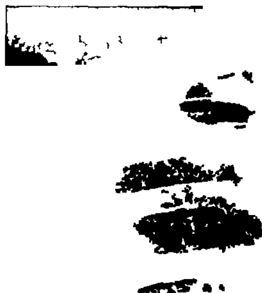


FIG. 36. The azygos lobe.

noted on the film Each zone should be compared carefully with its fellow on the opposite side before any condition noted is judged as a definite departure from the normal It has already been noted that disease processes are never exactly repeated in opposite zones and that if this s mple precaution is not employed it is possible for the common condition of calcification of the first costal cartilage to be interpreted as a tuberculous focus

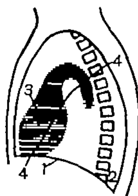
Normal lungs appe r as dark eas with many linear markings throughout them hue to the shadows of the main pulmonary arterial branches and to supporting structure of the parenchyma W have seen that the ha r-like line of the lesser fissure can appear in the third or fourth b interspac no other part of the pleura or its fissures should appear in an bsolutely normal film although it is not unusual to find an azygos lobe in the right upper zone Here fine line divides the right cupola by running downwards and inwards to the upper part of the cardiac border at which point there may appear a well marked but small oval opacity which is the azygos vein round which the visceral pleura has dipped during development This lobe has a light loss of trans lucency as it contains less residual air than the outer part of the upper l be

We ha already noted that the radiations due to the pulmonary arterial supply go outwards from each hilum which is itself due in most part of its confused shadow to the main arteries and the pulmonary veins Along their course and also lessening steadily towards the periphery we may see round opaque shadow of blood vessels caught end-on by the rays as they go backwards In the same w y while we do not see normal bronchi which go laterally we can see the round w ll-defined circles with clear centres which ar the shadows cast by backward going bronchioles (see Fig 35) They may be of fa r size near the hilum and hould not be read as cavities or mistaken for primary foci of childhood tubercle which are opaque irregular and hard looking and usually well out towards the periphery

At the danger of repetition it must be stressed that ariations in the normal striations are as much evidence of abnormality as the shadow of the disease process which causes the ariations If we see in the outer part of the right lower zone the shadow of pleural effusion we must look carefully at the striations in the mid and inner parts of this zone If the lung has no disease in it w expect to see heavier striations caused by hyperaemia from the

pressure collapse of the parenchyma If we can see no vessel markings ve know we are dealing with a pleural effusion which is secondary to a consolidation of the lower lobe

The Lateral Film



F G. 37 The left lateral film.

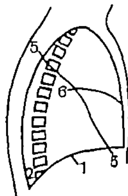


FIG. 37a. The right lateral film.

A lateral film may be taken to give additional information in the localization of lung disease For example it will throw a shado v that appears to be hilar into its true position in the apex of the low r l be in the case of abscess formation in that broncho-pulmonary segment v hich is supplied by the posterior horizontal branch of the right lower lobe bronchus

Lateral films may be taken of th right and left lung fields it must be realized that the hado vs of the opposite sid although out of focus lways contribute to the picture In th accompany ing diagrammatic representation of the left lateral film there can be seen between th vert b al column and the tern m —

(1) The diaphragm
(2) Th posterior recess of the costo-phrenic angle
(3) The cardiac shadow resting on the anterior surface of the diaphragm and continuous in its upper limit with the arch of the aorta

Th position of the main interlobar fissure is hown as a line (4) running from a point below and post rior to the apex downwards and forwards to a point near the juncti n of the middle and anterior thirds of the diaphragm The lower lobe therefore occupies the area below this lin

THE LATERAL FILM

On the diagram of the right lat ral film the position of the great fissure is shown as the line marked (5) while in add tion th lesser fissure is shown as (6), running between the hilum and the posterior border of the sternum It may appear on the lateral, as it does on the post ro-ant rio film n the absolutely normal subject

SECTION II

CHAPTER VI

PHYSICAL EXAMINATION—INSPECTION PALPATION AND PERCUSSION

PHYSICAL examination of the chest has suffered severe criticism in the past few years This is due mainly to the realisation among tuberculosis workers that phthisis can exist for months and even years *without producing symptoms and that in its earlier stages* it may give no abnormal findings to the most expert examiner whil showing definite abnormal shadows on the film The introduction of mass radiography was urged for these reasons its arrival has accentuated the criticism Nevertheless *physical examination like bacteriological examination must remain for* ultimate diagnosis The X ray is not the whole patient In the hands of an expert it can give detail of diagnosis with uncanny accuracy but without serial examinations it can seldom give the answer on activity or retrogression of the disease process The *clinical condition as a whole part of which can be summated only* by expert physical xamination can alone lead the physician to an opinion on the present pathogenesis on which he must base his immediate treatment

Nor must we confine physical examination to any one of its parts *Eyes first, hands next and ears last* is still a good guide It is the writer experience that many students concentrate too much on the stethoscope and that the statement that th re are no physical signs means usually that the examiner has found nothing abnormal to auscultation

It will be assumed that the student is familiar with all the usual methods only uch necessary aids and such points as the writer has found of h lp to post-graduates will be detailed

( ) **Clubbing of the Fingers** Inspection of the terminal phalanges should be mad in all patients suspected of chest disease, *acute or chronic* Clubbing ' is the phalangeal form of hypertrophic pulmonary osteo-arthropathy which is caused by the forming of a thin subpe iosteal layer of bone and thickening of

connecth tissue over it. It is found in chronic heart affections and chronic jaundice but is most commonly associated w th lung diseases Toxins and chronic congestion are said to have something to do with it but its real cause is unknown we do not know why in the terminal phalanges it show such marked thickening of the soft tissues in some cases which ha no apparent d fference in their lung pathology as against thei colleagues with no finger changes Its absence then is of no diagnostic signifi cance ts particular type when present can be so characteristic as to be diagnostic between septic and fibroid diseases between g lung abscess and pulmonary tuberculosis

The tw main types show first stage in common This con sists in a filling in of the connectiv tissue at the root of the nail so that there is no gap between the kin fold and the proximal end of the nail such as appears on the verage normal finger In addition, this excess tissue lightly raised and pushing up the kin fold, is always cyanotic and glistening, as if by ontinual polishing While this initial change is later in appearance i phthisis than in the acut septic conditions where as in int rlobar empyema complicating pneumonia it can appear within a few days of the onset it can be very helpful as a hint of the possible presence of symptomless phthisis

In the further hanges associated with proliferative tubercle of adult type the nail becomes humped bowed dorsally smooth and rather pearly-pink, while the palmar surfac of the phalanx becomes more and mo pendulous and baggy The finger is thus given a claw like appearance and may even look tapering as the dorsal-palmar alteration is not offset by any marked change in the lateral d mension

This is entirely unlike the effect of septic disease either acute or chronic as in lung abscess and bronchiectasis where the alter ation is generalized over the phalanx This alteration is in all dimensions gross and drumstick in effect often bulging sharply at the joint with the proximal phalanx, to stand out as an ugly blunt stump It can come on so rapidly in acute sepsis as to be noted in growth almost from day to day it can disappear as rapidly g after efficient surgical drainage of an interlobar empyema Naturally this form can lowly replace the more usual tuberculous type in long-standing fibroid cases where bronchiolectasis if not true advanced bronchiectasis is a common and e pected complication The patient in this case is suffering more from the

consequences of his disease than from present activity and bronchitis or bronchiectasis is all too easily given as a label to one who is a chronic source of tuberculous infection to all his contacts

In this connection we may note that emphysema is given as one of the causes of clubbing of the fingers From our consideration of pathology we shall see that emphysema is almost always a secondary cond tion. Its primary cause is the reason for the finger changes in the vast majority of cases

(*b*) **Inspection of the chest** as a whole should be made before any palpation methods are employed Much confusion can result in all examinations up to film inspection if the presence of the normal abnormality' of scoliosis is not recognized from the beginning A general inspection of the chest back and front should warn the examiner of the true condition, and keep him from making some diagnosis of pathological import It will prepare h m also for those changes from the absolute normal which he will see on the radiograph, in the shape and position of the heart, and ir the prominence of hilar and large pulmonary vessel shadows

PALPATION (*a*) The Sterno-mastoid Sign

Several methods of diagnosing mediastinal shift by assessing the displacement of the trachea have been described Most of these rely on deep palpation in the supra-sternal fossa a procedure to which most patients object very strongly A much easier and more reliabl method is that to which reference is made several times in the course of these studies It consists in gentle palpation across the tendinous origins of th sterno-mast id muscles at the uppe anterior surfac of the manubrium

W have seen in various chest conditions how the apex beat is not nearly *so reliable a guid to mediastinal* displacement how the heart swings on its long axis much more readily than it does bodily to one or other side and how for this reason while it responds to inspiratory movement as shown in screening of cases of atelectasis it does not give at positions of rest the same amount of fixed change to act as a guid to the examiner Thus it moves backwards (from left lower lobe emphysema) in right lower lobe collapse and its apex beat to palpation or stethoscope may give no aid to the actual diagnosis while the sterno-mastoid sign will be strongly positive on th right side

The main value of the sign i seen in cases of unilateral disease: t is only to be expected that bilateral disease will gi bilateral



FIG. 38. Positiv left sterno-mastoid sign

changes of similar type The onset of disease in the left side following an established disease in the right, will inevitably undo the value of the ign previously present in the right sterno-mastoid.

In acute conditions such as uncomplicated pneumonia, broncho-pneumonia and lung abscess, as in cystic disease of the bronchi and the epituberculosis of childhood it is negative We have discussed the reason for this These diseases do not destroy lasti tissue and have no effect on the normal pressure as usually exerted by residual air n the alveoli I chronic conditions with loss of elastic tissue telectasis and lat with fibrosis t is positive g in tuberculosis and bronchiectasis

Fo its discovery the patient sho ld be placed in good light, tanding, itting lying relaxed position with th head central to the thorax Gentle palpation with the index finger across the muscle tendon is q t ufficient the sharper edge of th affected tendon which as against ts softe rounder neighbour

will resist pressure is easily felt With very little practice the observer will see the sign before he applies palpation

(b) Lung Movement

Many students attempt to assess lung movement by placing the palms of the hands over the mammary regions with the fingers pointing upwards and outwards towards the outer sub-clavicular regions By so doing they cannot hope to find anything beyond the anterior forward movement of the thorax which is minimal as against the downward excursion of the lungs in response to diaphragmatic contraction and the lateral movement in response to the increased sub-atmospheric pressure of the intra pleural spaces The assessment of downward movement by percussion will be discussed below For proper assessment of lateral movement by palpation the palms and closely-approximated fingers should be placed gently but firmly in contact with the axillæ with as wide an angle as possible between the forefingers and thumbs The whole hands are then moved inwards still in close contact with the chest walls until the thumb-tips meet in the mid line of the sternum The patient is then asked to take in a long, slow breath, filling his chest without heaving up his shoulders Lack of movement on one side as against the other is immediately evident it *will not move the palm and fingers outwards* and the thumb on which these act as a lever will move but little from the mid-point as against its fellow If the method is properly employed expiration should bring the thumb-tips together again at the mid-point of the sternum The method is much more easy of application posteri rly

Where bilateral emphysema is present the thumbs move from the mid line hardly at all while the whole hands move upwards rather than outwards with deep inspiration

PERCUSSION

( ) **General.** The note in the lungs is individual and comparative one sid with the other The normal for any patient should be assessed on the finding at the left apex to gentle percussion, with the understanding that while sound enters into such assessment the feeling of resistance transmitted to the pleximeter finger by the underlying tissue is of equal importance Heavy tapping produces much noise but little to add to the knowledge of the

examiner Grades of departure from the normal can be described as impairment, dullness up to dead dullness tympany and marked tympany

A not on the left pex equal to that of the right must lead to a suspicion of left apical abnormality Its pictorial representation by one line helps us to compar It with mor marked abnormalities Thus two lines will represent the usual dullness of tuberculosis and bronchiectasis three the underlying resistance of pneumonia and fluid, and four the tonelessness and marked resistance of massive atelectasis and new growth Tympany is found in the higher pitch and lessened resistance of the basal emphysema of chronic bronchitis and marked tympany in the loss of lung tissue replaced by air in pneumothorax

(b) **Diaphragmatic Movement.** Percussion to be complet should includ examination of the change of note at the lung bases w th inspiration and expiration as by this means diaphragmatl excursion can be assessed A normal diaphragm hould mov downwards at least one finge s breadth n the axilla and one and a half breadths in the mid line of each half posteriorl with deep insp ration Lessening of extent of such movement w ll indicat adhesion up to complet fixture of the diaphragm Examples of the usefulness of percussion in diagnosis re —

( ) The dullness of upper lobe phthisis is predominantly post erior we ha noted sev ral times how tuberculosis in adults is posterior disease When theref re a decision has to be made on physical signs between upper lobe pneumonic phthisis and collapse from intra bronchial obstruction this finding is a help seeing the collapse w ll have its dullness mainly anterio and most marked bout one finger breadth below the inner third of the colla bone at which point w are ne the entrance of the epiarterial bronchus

(2)A collapsing right low r lobe will show dead dullness in th inner half of the right lower zone posteriorly offset by tympany from emphysema of the uppe lobe in the costo ph eni angle As against this lower lobe pneumonia show dullness anteriorly and posteriorly equal at any one lev l from the ax lla to the media stinum wh le the dullness of pleural ffusion w ll be axillary and that of collapsing right middle lobe anterio and most marked ust below the nipple

CHAPTER VII

AUSCULTATION

No student can hope to be able to distinguish the various abnormal sounds that can be found by auscultation unless he has accustomed himself to the many variations of the normal as they present themselves to his individual sense of hearing and tone. He should avail himself of every possible opportunity to examine healthy lungs in the fat the thin and the muscular using one uniform rule as his base line—e g always listening in the upper axilla or to the inner side of the lower inner border of the scapula, while the subject takes long quiet breaths through the mouth

He will soon find the following three characteristics of the normal —

(1) The sound is a gentle rustle as if made by an infinite number of individual sounds that run together an occasional one of these being slightly louder than its neighbours

(2) Inspiration is slightly longer than expiration

(3) Inspiration fades into expiration with no interval

If now he will listen by placing his stethoscope o er the trachea he will find four distinct changes

(1) There is a distinct gap between the end of inspiration and the beginning of expiration

(2) Inspiration and expiration are of equal length

(3) There is an entirely new quality in the sounds They are pitched higher than normal as if the normal sounds of a large area were concentrated at one point or the patient were blowing gently through a tube

(4) The sounds are louder

The first and second are present in every type of breath sound that is called bronchial breathing the third and fourth vary considerably in different disease conditions all of which are described as giving bronchial breathing

Naturally every teacher demonstrates his own acuity of hearing and sense of tone The writ r has used the f llowing rough rule of thumb —

I Ove scattered areas of fibrosis and atelectasis such as are

found in chronic pulmonary tuberculosis and bronchiectasis and over a larger single area of compressed lung (sometimes e.g. also a pleural effusion) we get the pitch as described without the loudness That is we have Pitch +

II Over a small area, e.g. over the point of occlusion of a bronchus filled with new growth or foreign material and over a larger area of consolidation, as in lobar pneumonia we have comparatively Pitch + + loudness +

III Over a cavity connecting with a patent bronchus we have Pitch + + + loudness + +

Here the pitch is so much higher that the listener has the sensation that his patient is now blowing over the end of a tube not through it

What now follows is built on the writer's thesis that chest diseases produce interference with normal breath sounds by giving extra sounds which can be reduced to a very few basic types and that what denotes the individual disease process producing them is the position of onset and intensity during inspiration This thesis is built on a conscious effort over twenty five years of observation to correlate intensive study of these sounds with the corresponding *ultimate diagnosis* It follows that its definitions and consequent statements will to many appear to beg the question, and to others to be dogmatic They are not meant to be so they are stated as the writer has employed them in his practice and has attempted to teach them to students

Every student should acquire the habit of listening intently to the whole phase of inspiration while the patient takes long quiet breaths through the mouth, so that noises of possible nasal obstruction do not interfere With the first breath he should try to assess the length of inspiration with a view to dividing it consciously if arbitrarily into three parts which we shall refer to as the bronchial, bronchial parenchymatous and parenchymatous phases The writer believes the student will be able to place abnormal sounds in accordance with their causative diseases as

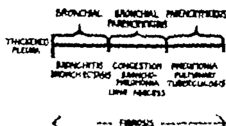


FIG. 39

these affect the bronchial and/or the parenchymatous structures. We hear the sounds known as sibili in both bronchitis and pulmonary tuberculosis but whereas in the former we hear them mainly in the first or bronchial phase in the latter they are mainly *in the last* or parenchymatous stage Thus the same actual sound can be of very differing import to the examiner and the patient. We see why *it is so easy* for a case of active tuberculosis to be interpreted as merely bronchitis

There appears to be considerable confusion in the minds of many students because they have no clear understanding of the true meaning of the many described abnormal sounds heard interfering with inspiration They are so subdivided in the usual teaching that they lead to confusion What is read by one physician as the only true sign of early tuberculosis may be read by another as evidence of a late stage of the disease

For all practical purposes the writer believes that added sounds can be reduced to three main types each with the particular significance of its underlying cause They are —

(i) Crepitations fine and coarse
(ii) Sibili and rhonchi (coarser sibili)
(iii) Râles fine medium and coarse (consonating or metallic).

I Crepitations

If the name crepitation be confined to a sound which has a dry quality to the ear we can correlate it with fibrosis i.e. increased deposition of connective tissue which we shall see occurs in various diseases considered under the section of applied pathology The breath sounds are now so changed that their individual components appear to be a series of dry crackles The coarser these sounds become the less seems to be their number They will indicate to us permanent structural change which is not removed by cough a restricting material which lies round the bron hi and attempts to interfere with their natural expansion and elongation in response to the act of inspiration In the pleural cavity fibrosis is the result of adhesion bet een pleural surfaces We can hea crepitations therefore at two points in our examination

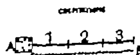


Fig. 40.
A. With act of cough in pleural thickening
B. With fibrosis of lung.

(1) First they can appear by the act of coughing They are

then fine approximating to the sound produced by rubbing the hair above the ears. We are hearing the disturbance of inter pleural adhesions which cough produces. They are a common finding in pulmonary tuberculosis and lead us to conclude that there is irritation of the pleura from an underlying lung lesion close to the surface. Naturally once the layers are firmly approximated and no longer at the "bread and butter" stage of organizing fibrin they will disappear. Their finding can be of significance in prognosis and in treatment, e.g. in considering the advisability of inducing artificial pneumothorax.

(2) The comparatively coarser crepitations of peribronchial fibrosis are heard throughout inspiration and do not disappear after cough. They are heard in all chronic diseases from chronic bronchitis to pulmonary tuberculosis as evidence of chronic hyperaemia and the consequent deposition of fibroblasts in the connective tissue. If we listen carefully we may be able to find by their commencement in one particular phase the part of lung structure on which the main brunt of the disease is falling. Thus if they are most insistent in the first or bronchial phase tailing off in the second and third phases we know we are listening to fibrosis round the larger bronchi involving surrounding lobules and that the patient has the way open to enlargement of these bronchi up to established bronchiectasis (see Fig 4). This is what experience shows occurs in cases of continuing upper sinus infections which cause constant irritation of basal bronchi.

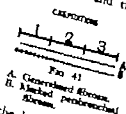


Fig 41
A. Generalised fibrosis.
B. Marked peribronchial fibrosis.

In the middle or bronchial-parenchymatous phase they indicate that combination of bronchial and lung fibrosis that occurs in lung because of a chronic character. The disease began in the lung. It is now infecting the bronchi that supply the diseased area of lung tissue and we are hearing the resultant fibrosis.

In the last or parenchymatous phase they are most intense in the presence of disease that begins in the parenchyma. Such fibrosis is general in lung where pneumonia has healed by organization rather than resolution. In the more localized areas of chronic adult phthisis it shows the sclerosis that has occurred in the lobular constituents of the affected parts.

Fibrosis in these three phases warns us of the danger of developing cavitation in each of them; it precedes the origin of cavitation

and remains when they appear As already stated the crepitations are indicative of a change that will not recede, that may be masked by emphysema later but may also lead to much more serious damage if the prime cause continues If we read into them the lessons they try to con ey they will aid us in prognosis as well as in diagnosis thus their continuance in the mid-phase after the apparent complete evacuation of a lung abscess warns us that our patient is not yet clinically well that a so-called recurrence of his abscess may follow and that what is really threatening is a consequent bronchiectasis by involvement of the bronchus

2 Sibili and Rhonchi

These are best interpreted as indicating moisture obstructing the passage of air through bronchi small and large While very little moisture in the smaller bronchi will produce sibili still smaller amounts in bronchi under contraction will give the same sound The quality of the sound does not differ by the origin of the obstructing material In other words, we can hear sibili that are due to stripping of the ciliated epithelium in acute bronchitis and sibili that are due to material entering bronchi from breaking down parenchyma in phthisis Again we hear them in acute asthma, because even the natural moisture normally removed by the action of the cilia cannot be dealt with the last circular muscular fibres are contracting so severely in th terminal bronchioles

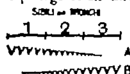


Fig. 42.
A. Bronchitis.
B Pulmonary tuberculosis.

If therefore we apply our scheme of dividing inspiration into three parts we can correlate sibili and rhonchi with their pathological cause

In the first phase they will be bronchial in origin they indicate bronchitis and bronchiectasis

In the second phase they vill be bronchial-parenchymatous We are listening to diseases which affect both bronchi and lung, *e g* broncho-pneumonia that is resolving, and lung abscess discharging In the first they will be over a considerable area of the lung in the second, they will be localized

In the third phase they will be parenchymatous in origin material is entering terminal bronchi from th lung usually from tuberculous deposits that are caseating Now the sibili and

rhonchi begin towards the end of the second phase and become mo and more intense to the end of inspiration

3. Râles

According to the thesis we a e f llowing the student should consider that râles mean that there is definite inv lvement of the lung parenchyma which is breaking down and discharging int a bronchus They indicat a greater amount of occlusion of the lumen of the bronchus than is present with sibili and rhonchi If they re especially coarse and consonating and metallic in charact r they should be ead as evidence of cavitation

By their position in inspiration w can get a definite aid to the underlying pathogenesis Thus if they are most marked in the first bronchial stage we can orrelate them with bronchial condition, and conclude they are extensions f a disease that has first produced a bronchial and later a parenchymatous pathological change That is why w hea them in bronchiectasis which has invaded the lobules through the penetrated bronchial lumen They herald the formation of a lung cavity connected with this as culation As the parenchymatous destruction continues they get more and more consonating and metallic in character Their finding is then diagnostic of bronchiectatic lung cavitation

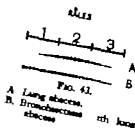


Fig. 43.
A Lung abscess.
B. Bronchiectasis ith lung abscess

In the writer experience coarse consonating metallic râles ar a more constant finding than whispering pectoriloquy in all types of cavitation They can help towards diagnosis when the cavity is comparatively full of disintegrating material and pectoriloquy and loud bronchial breathing may be absent

F ne râles ar heard in the bronchial-parenchymatous middl phase of inspiration in congesti e failur They indicat an inability of the patient to mov lobula moistur they are the first signs of that water logging which increases at ad ly in the chronic bronchitic of grav prognosis

Resol ing broncho-pneumonia gives them in this phase in medium intensity they come through to the ear almost as ind idual small bubbles rising n number

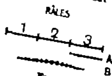


Fig. 44
A. Pneumonia.
B Bronchopneumonia.

and force to mid inspiration and then falling off again toward the end of inspiration We are hearing discharging lobules pouring their liquefying pneumonic exudate into the bronchioles

They are still more insistent and coarser in the discharging lung abscess although they are confined to one area as against those heard in the disseminated disease of broncho-pneumonia They become more and more consonating and metallic when bronchial blockage has led to breaking down of the lung substance *in a case now going on to chronic abscess We hear now exactly* the same sounds we heard in bronchiectasis There we heard them begin and have their greatest intensity in the first or bronchial phase and they were heard over a fairly large area of the lung Now we hear them in a localized area and most intense in the mid phase of inspiration The same pathological process is at work in both conditions In bronchiectasis the disease progressed from the bronchus into th parenchyma in lung abscess it progressed from the parenchyma to the bronchus Chronic lung abscess is only a localized form of bronchiectasis

In both cases as the disease progresses the râles get more and more metallic in type telling us that communication between cavitation in the lung and an enlarged bronchus is now established

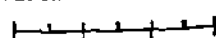


FIG. 45 Metallic râles in tuberculous cavitation.

In the last, parenchymatous phase we find them in pulmonary tuberculosis With an advancing lesion they slowly replace the sibili and rhonchi of lesser obstruction while like them they are due to material ent ring the terminal bronchioles they are now due to *actual* breaking down of the parenchyma As the condition goes on to established cavitation the râles get more and more metallic, reaching their crescendo of sound at the ery end of the inspiration

The scheme of interpretation of added sounds is summed up in Fig 39 which the writer has found useful in teaching on hospital cases for practical demonstration

Two examples will illustrate diagnosis from physical signs alone —

(1) *Chronic fibrocaseous tuberculosis with cavitation of the right lung upper half*

Clubbing of tuberculous type is present in most cases The right sterno-mastoid is strongly positive There is marked lack of movement over the whole of the right side dullness in the upper

half especially posteriorly and tympany from emphysema over the lower half

Careful listening to the whol inspiration ill present crepitations throughout every phase becoming more and more coarse in the third phase these are from fibrosis At the end of inspiration there will be coarse metallic râles from the cavitation Both crepitations and râles ill remain and may be even more insistent afte cough With the act of oughing showers of fine crepitations indicate pleural thi kening

(2) Acute lobar pneumonia right low r lobe before resolution There is no clubbing of th fingers There is no terno-mastoid sign There is marked lack of mo ement ll over the right id and dullness both anteriorly and posteriorly er its low half There are no added sounds th breathing is bronchial all over the affected lobe

SECTION III

CHAPTER VIII

SOME GENERAL POINTS AND THE VISUAL, OBJECTIVE DESCRIPTION OF THE ABNORMAL FILM

In this section we shall gather together such changes from the normal as we shall see in the films of diseases discussed in detail in the section on applied pathology It is possible to group them under their main characteristics For example most diseases of the lungs have in common an inflammatory exudate filling lobules as a result of irritation of their alveolar walls this common process has a common homogeneous X ray shadow It is denser than that of thickened plastic pleurisy but far less dense than that of atelectasis It therefore follows that the shadows seen in these three separate conditions are all objectively described as homogeneous, although they may by the experienced eye be correlated with their individual underlying pathology especially if they are studied in conjunction with the clinical details These should always accompany the request for a report on the film of a patient who has not been clinically examined by the reader In other words a film report should be in two parts the first a visual objective description of what is actually seen the second an opinion on the cause

It goes without saying that anyone attempting to give an opinion on an abnormal film must have an extensive knowledge of the normal and of all normal abnormalities Reference has been made several times to difficulties that can arise by the reading of a pathological cause into the normal abnormalities of scoliosis, or simple calcification of the costal cartilages Even the most experienced observer does not despise the assistance of a good example of the normal

THE VISUAL DESCRIPTION

Some general rules for guidance may be set down —

(1) Read every film according to a fixed routine such as that given in the chapter on the normal film

(2) In reporting n the trachea, h art and diaphragm not any change in size shape and positio

(3) In th lung zones look first fo th essel markings and the lesser fissure of the right lung, noting any increase decrease or change in position such as bunching o splaying of ascular shadows

(4) Describe abnormal shadows according t their basic types remembering that in front of them behind them there may be shadows of normal structures They may be considered und five basic types streakiness homogeneous shadows non-homo geneous shadows ring shadows and miliary shadows

Streakiness is the name pplied t increases in the normal lung markings

Homogeneous shadows are those which do not alte in their evenness throughout thei extent lthough they may lte in density they do not d so suddenly and interruptedly

Non-homogeneous hadows are those that alte suddenly in density at one more points throughout th i extent

Ring shadows ha more o less circumscribing limits In thei centres they may ha homogeneous o non-homogeneous shadows areas of translucency Similarly thei limiting boundaries may consist of one ther f these types f shadows These hadows will be described in more detail in the next chapter

(5) Give the size and shape of individual shadows they may be singl m ltiple large o small well ill-defined diffuse along th lines f normal markings confluent r patchy round oval o triangular

The term ' mottling is often used in descriptions of shadows It conveys th ide f mixture f opaque and non-opaque reas In this sense it can apply to such shadow as occur in th film of uncomplicated broncho-pneumonia It is however often used to describe a mixture of homogeneous and non-homogeneous areas such as are found in the film of pulmonary tuberculosis and some interpretations confine it to this meaning It is theref re not a good word to use as being in the literature half descriptive and half interpretati and with no accepted definition under either heading

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Ring shadows ha more o less circumscribing limits In thei centres they may ha e homogeneous o non homogeneous shadows o areas of translucency Similarly their lim ting boundaries may consist of one other of these types f shadows These shadows will be described in more detail in the next chapter

(5) Give the ize and shape f individual shadows they may be ingl multiple large or small well ill-defined diffuse along the lines f normal markings confluent r patchy round oval triangula

The term ' mottling is often used in descriptions of shadows It conveys th idea of a mixture of opaque and non-opaque reas In this sense it can apply to such shadows as occur in th film of uncomplicated broncho-pneumonia. It is however often used to describe a mixture of homogeneous and non-homogeneous areas such as are found in the film of pulmonary tuberculosis and some interpretations confine it to this meaning It is therefore not good word to use as being in the literature half descriptive and half int rpretativ and with no accepted definition unde either heading

CHAPTER IX

THE INTERPRETATION OF ABNORMAL SHADOWS. STREAKINESS HOMOGENEOUS AND NON-HOMOGENEOUS SHADOWS

BEFORE giving an opinion on the diagnosis of a patient under review the examiner should now inspect the film more closely at about 1 foot from the viewing box and then go back slowly to a distance of some 5 or 6 feet The writer realizes that this will not make the film stereoscopic what it will do is to make individual more opaque areas within a shadow that looks more or less homogeneous on close inspection stand out against any background common to them Unfortunately this point cannot be demonstrated by prints but only by the study of films on viewing boxes

There will always be the difficult cases where shadows are so confused or ill-defined that their underlying pathology cannot be diagnosed There is no such thing as the typical film for every disease there are always individual differences Furthermore, we all understand that X ray examinati n is at best only an accessory to general and specialized physical and laboratory investigations. Consideration of the general condition, the temperature the blood-count cellular content of effusion and the result of bronchoscopy and biopsy may have to come into the summation of the interpretation together with serial film examination, before we know in any one hypothetical case whether we are looking at a lobar atelectasis which is basically post pneumonic post-operative or due to carcinom of the bronchus There are, however from the experience of various disease conditions as reviewed under applied pathology certain guidances to be had if we look now more closely into the main types of shadow defined in the last chapter It is understood of course that while these are read individually their accompanying shadows and the evidence of interference or non-interference with other tructures of the thorax are read with them for interpretation Thus the shadows of pneumonia and of telectasis of the right upper lobe are

both homogeneous in type but we ha seen that there is no change in the blood supply f the middle and lower lobes in pneumonia, whereas in t lectasis there is splaying f the r blood supply since they have become mphysematous

Streakiness and Absence of Normal Markings

Decrease and increase of normal lung markings are equally important Decrease is found in pneumotho ax cystic disease of the bronchi and emphysema In pneumothorax the loss of markings appears outside the line of the collapsed lung, while this is offset by increased markings within it W th cystic disease some faint markings of normal lung in front of or behind the cyst may occasionally be seen but usually there is ircumscribed area of the translucency of pneumothorax Often there are with larger cysts definit increased markings outside the cyst due to pressure collapse of surrounding lobules nd the crowding togeth r of their normal struct ral connective tissue and blood supply

With emphysema there is decrease o small or large a eas of the lung, gi ing an increased translucency of the affected part Where considerable portion is affected as in th gross basal emphysema f chronic bronchitis or in the osto-phrenic angle emphysema f the upper lobe d e t lower lobe telectasis only the faint markings of the supporting structure of much-distended lobules can be seen Such film diagnosis of emphysema may be of great import to the patient As lready stated if t is not accompanied by apparent X ray cause t ordinary film examination it may call fo special examination by lipiodol injection or bronchoscopy

Increased streakiness is found in hyperaemia We hall see how hyperaemia is the first ind cation of acute bronchitis and the constant accompaniment if not the bas cause of chronic bronchi tis W hav seen too that w ought t look for hyperaemic markings in a lung und r collapse from pleurisy w th ffusion and how w must suspect consolidation of th lung if w do not see them

The distinctive feat re of all shadow connoting fibrosis is that they stand out n increasing relief if we bserv th film as we go backwards to some 5 feet from it This characteristic they share with those shadows w have named non-homogeneous In chronic bronchitis they are almost invariably along definit lines as indicating that there is increase in perib onchial connective tissue caused

by the hyperæmia of its vessels With fibrosis we may see corroborative evidence of the true pathology in disturbance of the normal position of other recognizable structures e g change in the position or density or both in the lesser fissure rise of the diaphragm and perhaps displacement of the heart and mediastinum in such a disease as bronchiectasis Again we see streakiness perhaps dissociated from the usual normal markings in carcinoma, especially in the nodular medial and basal types We find it also with or without discrete nodules in the reticulations of silicosis when we are viewing the congested and thickened lymphatics associated with this disease

Homogeneous Shadows

These shadows when in the lung result f om exudates and de-aeration in the pleural cavity they are due to exudates and occasionally to transudates

On the whole it can be taken as a safe rule that homogeneous shadows indicat acute lung disease They are found in pneumonia, broncho-pneumonia and in the acute progressive parts of tuberculous lesions Their variation in density can gi us a hint of their underlying pathology Homogeneous loss of translucency of lesser density is found as the X ray evidence of inflammatory reactio i in the alveoli This is due to irritation of the walls of the cells by the organisms which later penetrate the walls and fill the cells with exudat Such exudate throws a denser but still homogeneous shadow

It follows that und r this type of shadow falls that called by the name of pn umonitis This term is justifiable if it is merely descriptive and not meant to be specific and interpretative It is not a pathological entity It is the shadow common to every process irritative to lung parenchyma being the œdema or catarrhal exudate seen in pneumonia, broncho-pneumonia lung abscess, bronchiectasis and tuberculosis Its presence means that some specific disease is t work, g it may be the shadow of fleeting type such as we ee in tropical eosinophilia In every case we require serial films and we may require lateral and t mograph examinations and even lipiodol injection or bronchoscopy to reveal the underlying cause

In uncomplicated pneumonia and broncho-pneumonia we see a homogeneous shadow involve the broncho-pulmonary areas of a main bronchus or one or more of its branches In phthisis we

see it in the localized reas of dorsal bronchioles In both we can follow its progress from the catarrhal to the specific heavier shadow so that we are now able on the film to recognize the ad vancing edges of the disease process Sim larly we may note the first loss of translucency in telectasis which comes w th the absorption of oxygen and trace t to the last which is due t the actual invasion of the parenchyma by carcinomatous moem This last and densest stage is most evident sometimes from the beginning at the site of bronchial blockage

On the pleura we recognize the first generalized fine loss of translucency that comes w th plastic pleurisy so often the back ground to a tuberculous lung lesion to an underlying lung abscess. The same fine loss of translucency is found in the shadow of the opalescent ffusion of tuberculous origin increasing n density with the organization of fibrin throughout it and n especial on the visceral layer of the pleura against the lung which is under pressure collapse If the ffusion becomes loculated and begins to organize in the pleural cavity r becomes purt t gets more and more opaque until it reaches the opacity of pneumonic exudat in the lung We ha then the density of the shadow of empyema

All homogeneous hadow share one particular characteristic whether they be due to lung or pleural pathology they do not alt r in their und rmity when iewed at a distance The shadows of pneumonic exudat n pneumonia broncho-pneumonia and lung abscess, are homogeneous in this sense The wall of a proliferativ tuberculous ca ity w hall see is never homogeneous W th two examples we hall be able to illustrate this point more fully

In broncho-pneumonia w an f llow the progress of the disease in shadows first evident near the upper halves of the right auricular or left ventricula bord rs of the heart, going then outwards and downwards along the lines of the main blood supply to the lower lobes In the developed lesion w recognize along these lines, homogeneous triangular like areas of equal loss of trans lucency nev r quit discrete but never quite confluent because between them re areas of inner homogeneous loss of trans lucency The first shadow is due t pneumonic exudat the second to the non-specific inflammatory catarrhal exudat Neither changes in type or sharpness by distant as against near viewing This is because w are looking at material in the lung lobules that have not organized There is no collapse of lung cells We have corroborati evidenc in the absence of mediastinal shift

CHAPTER X

ROUND SHADOWS

RING shadows may circumscribe areas of comparati density or comparati e translucency The ring f rmation may be of homogeneous or non-homogeneous type whethe t is thin or heavy in outline

( ) **Ring Shadows round Areas of Comparative Translucency** The thinnest of all homogeneous boundaries is t und in emphysematous bullæ as here it consists only of the visible strands of stretched supporting tissue round an area in which no connective tissue streakiness is seen ther because normal tissue is tretched or broken beyond recognitio because the tissue is absent by congenital abnormality We se such areas particularly tow rds th upper and uppe -out portions of th upper l bes and along the infer and lower ant r margins of the lower lobes which areas are naturally weak in connecti tissue W recognize these bullæ sometimes in the lung f a child who has had se ere whooping cough oft n in the chroni bronchitic towards the bases and occasionally in th ongenital and so-called essential emphysema f unknown recognizable rigin.

These bullæ do n t look in anywise different from type of ring shadow sometimes seen in known cases of pulmonary tuberculosis the writ has watched many of them come and go in that type of case whi h seems t ha e more than rdinary pleural reaction to the und rlying disease Some disappear altogether as quickly as they appeared but many lea in th ir low zones ragged fibrotic looking shadow which now produces in previously silent area those dded sounds which we associated with pleural thickening; burst f fine crepitations with the act of coughing They would theref re ppear to be the localized emphysematous bullæ localized pneumothoraces ove small lesion the lung surface

A more visibl but till homogeneous outline rounded rathe than oval is seen in congenital cystic disease of th bronchi These cy ts may be ingle o multipl separate o apparently

which shows that these materials are replacing the residual air and not altering the normal pressure which residual air maintains.

Again in the film of adult proliferative phthisis we have, at some stages of the disease when exacerbations are taking place, the possibility of three homogeneous shadows First there is a generalized fine loss of translucency forming a background to all other shadows this is the plastic pleurisy of slow irritation and adhesion of the overlying layers of the pleura Second we see scattered areas on the edges of the main shadows they have a density slightly greater than the first, and equal to that of the catarrhal exudate of broncho-pneumonia Next we see the denser homogeneous shadow of tuberculous exudate in lobules where the tubercle bacilli have invaded bunches of acini to produce true tuberculous exudate None of these alter materially on distant viewing while all three incline to merge when we look very closely The distant view will however throw into strong relief any non-homogeneous shadows also present

Non-homogeneous Shadows

These are shadows which alter materially in their density within their extent on viewing at ordinary distances and still more so when viewed at a greater distance As against homogeneous shadows which mean acute lung disease we can take it as safe rule that they mean chronic disease—bronchiectasis and pulmonary tuberculosis being the commonest In other words, we can correlate them with fibrosis and organization, and with the type of atelectasis which results from these two processes. They are denser more clear cut and harder looking and usually very irregular in outline They may on close inspection appear to be confluent with the rest of the abnormal shadow of which they are part but on distant viewing they stand out sharply This is because they are surrounded by lobular complementary emphysema the continual accompaniment of atelectasis in all its forms We have already noted that there can be no collapse in either small or large areas of th lung, without corresponding emphysema The translucency of these emphysematous lobules acts as a foil it offsets the o ganized areas It is therefore possible to writ an interpretative report of tuberculous infiltration, fibrosis and pleural thickening

more common in the lung abscess which is not evacuating freely because th bronchus connected w th t is block d

(b) Round Shadows of Areas of Comparative Density

An abnormal shadow may be rounded re of loss of translucency of comparati dens ty

The Assmann's focus l k lung bac st is f und t those particular segments supplied by d rsal bran h of the bronch It is homogeneous rathe fluffy n t dges but on distant wing stands out as a whole against its rround ng t ssue O serial





F 46 Assman focus in right upper lobe

films it may emain unchanged fo onside bl pe d When it breaks down t occasionally doe rs qu kl t n sho an exceedingly lear ent usually ho t loses ll semblance to round shadow be m ng dragged ut t w ll into the exaggerated blood essel markings of the inflamed b onchi long which the ontents f the now nfected pa enchyma a ttempting to drain to the glands A distingu hing f ature h h

superimposed (see Fig 9) They may have walls varying in thickness but seldom of more breadth than a 1/16-inch on a 15-by 12 inch film They may contain a fluid level If connected with their originating bronchus they can share in bronchial affections up to the sepsis of bronchiectasis and can sometimes be recognized in a collapsed lobe Ordinarily however in their uncomplicated state they have two definite characteristics in addition to those stated which can aid us to give an interpretative reading First they have no associated evidence of fibrosis round them second they have no associated mediastinal shift

The outline of lung abscess which is discharging gi es the next more evident homogeneous ring shadow enclosing an area of comparati e translucency The boundary is now round ¼ to ½-inch in depth It usually has a generalized hazy loss of translucency external to it and over its enclosed lung tissue this being due to thickened pleura and on its outer edge to catarrhal exudate in those alveoli which ha e been irritated but not actually involved in the disease process The actual boundary is made of pneumonic exudate and so does not alter materially in whole or in part with distant vie ing as against closer inspection It has no surrounding fibrosis It is therefore in every way a single entity in the lung field o al rather than rounded Its homogeneous character tends to remain up to its change to the secondary stage of bronchiectasis, its inner pneumoni constituent appearing to be frozen *in situ* even while its outer lining show increasing and decreasing densities with advancing invasion of its sepsis int the surrounding lung Moreover it still remains an entity and not just part of a generalized invol ement of the particula lobe or of the lung field in which it is situated as is nearly always the case with the tuberculous ca ity Apart from the acut exca ation of an Assmann s focus a tuberculous cavity is seldom without definite surrounding fibrosis

The average tuberculous cavity can b visualized from what has been already described (see Fig 103) Its urround in both internal and xternal borders is irregular and uneven It merges in its outer zone with evidence of fibrosis This fibrosis with organized and collapsed lobules that are fused together and opaque form its wall which stands out in bold relief to distant viewing It is seldom the only evidence of disease in the lung It may contai a fluid l el which shows as a homogeneous shadow with a straight horizontal upper border but a fluid level is much

formation It has much more paque t nt duc t l er l filled with pneumonic exudat it edn lu lu los d t nluc ncy due to catarrhal exudate n surr u l ng l l hul k ly fades into normal tissue shadc n A lnc nd gr g pon taneous evacuation is not lea n t t n the udat d na int the bronchus along hi l (as hald l h k fading from the centr of the hadc n t n nl the l u d The n ll



FIG. 48. Lung Abscess

theref r be tage bel disappearanc of ll bnc rmal shadow ng when the catarrhal haz rcuml renc has disappea d and the central heavy homogeneous opac ty has been replac d by loss of translucency whi h becomes graduall dense f om the ent towards a f ly defined ring shadow This ring w ll lso be homogeneous as it is cast by alveol which are t ll filled w th pneumonic exudate The d ff ntial diagnosis of hronic becess and tuberculous ca itation is discussed later

A hydatid cyst is not a finding although primary are ra er than secondary cysts The shadow f the individual primary cyst is homogeneous ry dense clear in outline and

will therefore be the associated fibrosis in and around its supplying bronchus

The Assmann's focus is therefore never like the cyst in congenital cystic disease which is affected by acute bronchitis the cyst does not stand out so clearly against its background and as the infection subsides its wall returns to the characteristic thin outline with no resulting fibrosis round it

There is a type of nodular carcinoma which starts in a small bronchus and involves the parenchyma through the bronchial wall to produce a single rounded shadow It can resemble an Assmann's

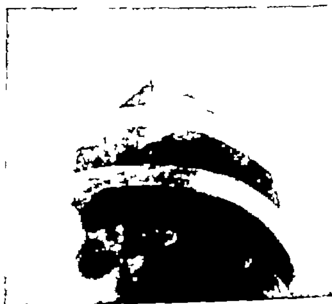


Fig. 47 Nodular Carcinoma.

focus so closely that it may be impossible to diagnose it with the fullest radiological examination Only serial films may eventually give the true diagnosis as happened in the case illustrated Usually however such a shadow is basal and though it is mainly homogeneous is much more dense than the Assmann's focus and has definite streaks connecting its inner border with the hilum

An acute lung abscess, often oval rather than rounded has not the clear outline of the Assmann's focus and does not stand out against its background on distant viewing Seen just after its

The hydatid will be found to collapse with lung tissu while the others retain their previous relati e position to th mediastinum and the bony thorax

A large calcified tuberculous focus is unlikely to cause d fficulty as calcified hilar glands will be evident

Serial films will differentiate against an Assmann focus o a lung abscess During the period when we expect changes in the hazily outlined abscess and in the lea -cut Assmann s focus which began by standing out against its background on distant lewing th hydatid will cast the same dense homogeneous and rounded shadow

This sam characteristic of remaining static in size and density for considerabl period will d in diagnosis of multipl hydatids against the shadows of metastati growths if these are confined t the right lower lobe They increase density with age d ffer in size and grow steadily Usually they in large numbers which is diagnostic feature The miliary appearanc they can prod when they suddenly appear in both lungs is considered n the next chapter

The conjunction of tuberculosis and diabetes can gi many round shadows Two points d in diagnosis they stand ut well on distant lewing against surrounding mphysema and they are confined to the upper and mid zones

right lung and in its lower lobe It *can vary considerably in size* Naturally if hydatid cyst is suspected precipitin complement fixation Casoni and blood tests must be done the majority o cases will gi e a positi e Casoni and an eosinaphilia

Multiple secondary hydatids occur usually in the right lower lobe and as they are common results of rupture of a sub-phrenic abscess the li er must be examined and special attention paid to the shadow of the right diaphragm this shadow will often be raised or dome-shaped or indefinite in outline

As against the singl hydatid we may ha e to consider the rare true benign chondroma and the occasional neuro-fibroma and dermoid which appear to be in lung tissue This is because all



Fig. 49 Dermoid of Lung.

three may be rounded clearly defined dense and homogeneous. While the chondroma goes on to hard entral areas which are very d nse by ossification of th cartilage remnant from which it dev loped as against th peripheral density of the hydatid *It may* be necessary for differential diagnosis to induce pneumothorax

The hydatid will be found to collapse with lung tissue while the others retain their previous relative position to the mediastinum and the bony thorax

A large calcified tuberculous focus is unlikely to cause difficulty as calcified hilar glands will be evident

Serial films will differentiate against an Assmann focus or lung abscess During the period when we expect changes in the hazily outlined abscess and in the clear cut Assmann focus which began by standing out against its background on distant viewing the hydatid will cast the same dense homogeneous and rounded shadow

This same characteristic of remaining static in size and density for a considerable period will aid in diagnosis of multiple hydatids against the shadows of metastatic growths If these ... confined to the right lower lobe They increase in density with ge d fi n size and grow steadily Usually they are in large numbers which is a diagnostic feature The miliary appearance they can produce when they suddenly appear in both lungs is considered in the next chapter

The conjunction of tuberculosis and diabetes can give many round shadows Two points aid in diagnosis they stand out well on distant viewing against surrounding emphysema and they confined to the upper and mid zones

CHAPTER XI

MILIARY SHADOWS

Numerous or uncountable areas of loss of translucency can appear in certain or in all zones of the lung fields Interpretation of the under lying cause depends to a very great extent on the quality of the film especially on its penetration This is one reason why there is still such variance on the diagnosis of reticulation, which may be read as present in a soft film and be entirely absent on a hard film Any film of any diseased condition is necessarily like the film of a normal lung a photograph of superimposed structures so that we may be quite unable to differentiate miliary shadows unless we know the patient s past history and in particular his occupational history Indeed without serial examinations we may even then fail to make a satisfactory differential diagnosis as between miliary tuberculosis silicosis and siderosis With this caution, however we may find it possible to give a reasoned opinion if we take the following five criteria in conjunction the predominant distribution the size and shape of individual shadows the density of these shadows their type of background and the clarity with which they stand out against that background

Only some of the more usual conditions will be considered Such conditions as byssinosis siderosis and asbestosis are too specialized to warrant discussion here

Acute Miliary Tuberculosis

The acute form of miliary tuberculosis gives a standard against which other conditions may be considered Both lung fields are covered in every zone from apex to base with dots which vary in size and shape because they are photographs in different planes of the catarrhal reaction to microscopic deposits of tubercle They are usually at most three millimetres in their largest axis Individual shadows hav a light loss of translucency but are often so cro vded togeth r that no lung markings are visible with the result that their background appears as a dirty off white discolour-

tion. This background da kens steadily on uccess films wh the individual deposit coalesce in some r as t ppea as mer ill-defined smudges ith superimposed f t



FIG. 50. Acute miliary tuberculosis

Chronic Miliary Tuberculosis

The chronic f rm i its ea ly tages may d feat d ffe ential diagnosis as against th acut f rm on ngle film xamination alone Its usual age incidenc of adc lescenc and ly adult lif and th latency of the disease must be taken into consideration Of patients who survi some show ppa ent learanc of ll the lesions others show gradual lessening in the number of deposits those remaining being scatt red in all zones but tending t show preference f r the upper and mid zones They re dense and sharp in outline U ually the background is mor transluc nt than normal but there may be increased striations of fibrosis round both hilar regions and over the upper pices In several

cases there is marked enlargement of hilar glands usually more evident on one side This stage of the disease is better described as calcified miliary tuberculosis



F 31 Healed miliary tuberculosis.

Bronchiolitis.

This condition is a blockage of the fine bronchioles as a result of their inflammation It can occur in children with measles or at any ge as complication of influenza or of poisoning by irritant gases such as phosgene and chlorine When the blockage is complete there is a resulting collapse of bunches of air cells which they supply If the air cells re-expand as a consequence of the clearing of the bronchioles no ultimate damage to the parenchyma will result It may be that in some cases in children the recovery is more apparent than complete and that this produces a cylindrical enlargement of bronchi which is later interpreted as congenital in origin Where measles is the prime cause the majority of cases how a marked coincident enlargement of hilar and bronchial glands

The predominant distribution of th shad ws is in the lower zones tailing off distinctly int the mid and upper zones even in extreme cases There is a im larity to the film f cut m liary

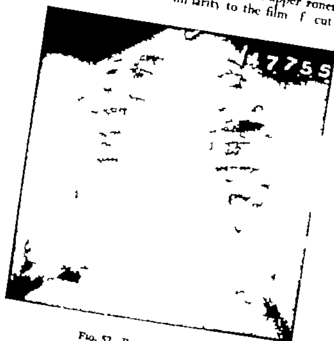


Fig. 52 Bronchiolitis

tuberculosis in the loss of translucency and indefin t outline of individual shadow but careful inspection how that they tend to be larger and to coalesce because bunches of alveol ar involved in the process The oc luded bronchiol can usually be distinguished as ery fine but defin t dot in th centre of the bunch By film alone the condition may be mposs ble t d ff rentiate from the results f inhalation of paraffin particles from long-continued nasal med cation f om th finding in cases f ship-wreck survivors who hav inhaled oil All three conditions usually clear up over short r longer periods

This is not the case with bronchiolitis obliterans where w see a permanent condition onsequent on th inhalation of irritant gases Inflammation has proceeded t fibrosis within the bronchioles which do not ecover Blast injuries during the war produced

an exactly imilar picture Bronchiolectasis is a very common end result The distribution i imilar to that al eady described,

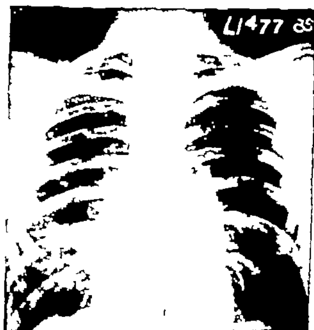


FIG. 53 Same patient as Fig 52 six months later

but indi idual hado vs are much more clear cut hil permanent organization of th xud t gives a more opaque shadow in which we can find no ce tral dot

Broncho-pneumonia

Broncho-pneumonia is unlikely to cause difficulty owing to its predilection for the lower zones to its tendency to follow the

lines of th bronchi and to its comparatively rapid changes on serial films between the hilum and the periphery



FIG. 54 Bilateral lower lobe broncho-pneumonia.

Silicosis

Medical l terature has contained many discussi late on reticulation which is stated t be the first stage of L exposure to small amounts f silica causes irri tearing f the lymphatics which re therefore t liminat it The result is blockage of, and this appears first t the bifurcation The film now shows a fine fibrosis

dots of silica accumulated at the bifurcations Experts say that it is the composite photograph of these dots taken in superimposed planes of the lung which can give on a soft film that first abnormality kno vn as r ti ulation

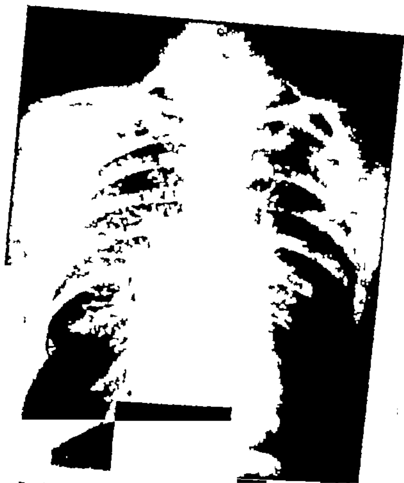


FIG. 55 Reticulation and early coalescence in haematite miner

When definit miliary hadows ppear dense and well defined nodules are seen standing out clearly against a background of

emphysema Such tage is occasionally reached quit rapidly so that there may be xtreme d fficulty in differential diagnosis as against one stage of hronic or of a calcified miliary tuberculosis.



FIG. 56. The nodular stage in haematite miner

Serial films w ll then gi th answer because th nodules will enlarge rapidly to ize much beyond that of tuberculous deposits and will coalesc i t irregular densit es Round these w ll appear much fibrosis and marked emphysema and individual nodules may be no longe recognizabl

In the usual course of the disease distribution of nodules is a good guide to diagnosis They first appear in the periphery of the upper lobes As they gradually increase in number they appear more and more towards the upper mediastinum in the posterior surfaces of the upper lobes and in the posterior apices of the lower lobes As we have already noted they have a predilection for the same dorsal branches of the bronchi as the deposits of phthisis

Anthracosis

Anthracosis or coal miner's black lung is a condition closely allied to silicosis indeed it may be extremely difficult to differentiate between them By long exposure to small amounts of silica

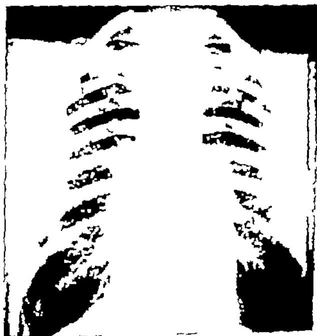


Fig. 57 Anthracosis, early stages.

in the coal dust the blockage we have noted in silicosis takes place at the bifurcation of the smaller bronchioles Beyond this point carbon accumulates until it fills and later distends the surrounding air cells When present this localized distension which can be

up to some five millimetres in diamet gi es a characteristic film Density of individual shadows varies consid rably some nearest the film appear quit hard Apical and basal emphysema is said to be a charact ristic finding



Fig. 58 Anthracosis with typical emphysema.

Mitral Stenosis

The appearance of m liary shadow n mitral stenosis is of grave significanc as they re idence of a failing heart Ind vidual shadows re rounded and homogeneous l the loss of translucency Close inspection of th film will how that they lessen st adily in every direction from the h lum to th periphery because they are the shadow f blood essels een end-on Th h lar shadow are confused and sw llen and ontal an oc as onal dense shadow thrown by th principal blood essels The ma k d loss f trans lucency in th mediastinum gradually merges into generalized lighter loss of translucency due to heart-failure lls which re filling the al eoli

The condition may cause difficulty as against one stage of a miliary tuberculosis or occasionally of an early pneumoconiosis



FIG. 59 Advanced mitral stenosis with miliary shadowing.

but the dense hilar shadow and the gradation in size of individual shadows which are less clear-cut in outline because of their dark background will generally help in differential diagnosis

Malignant Metastases.

Blood borne metastases of miliary form from a primary growth outside the lung are rarely seen The individual shadows are usually ill-defined blotches of irregular size and density Occasionally they can resemble miliary tubercles or the nodular stage of pneumoconiosis but they are usually more ragged in outline tend to increase more rapidly in size and to concentrate from above downwards so that the apices are comparatively clear Their sudden appearance and their failure to stand out on distant viewing because they have no background of emphysema, will also aid in differential diagnosis

While this form is practically unknown as a result of primary lung malignancy a growth may remain unsuspected in one of the

larger bronchi until it has reached the thoracic duct through the glands and flooded all the pulmonary lymphatics with malignant cells The lung then appears to be covered from apex t base by a loosely woven fine hair net This is du to thickened lymphatics where they cross or ppear next the film end-on w can see tiny nodules If a large mediastinal gland is seen this will h lp m diagnosis The condition is known as lymphangitis arcinomatosis

SECTION IV APPLIED PATHOLOGY

CHAPTER XII

BRONCHITIS AND EMPHYSEMA

Acute Bronchitis

In bronchitis both acute and chronic there are two changes from the normal of which we must take particular note These are increased blood supply and more or less destruction of the inner constituents of the bronchial wall The increased blood supply contributes to our film findings We have noted several times that the branches of the bronchial tree are closely associated with those of the blood vessels and that the latter show on a normal film In bronchitis these markings become more evident, being due at first to the congested blood vessels and later to increased connective tissue round the bronchi as a result of this hyperaemia

In acute bronchitis congestion causes swelling of the mucosal coat while all th coats become infiltrated with large numbers of polymorphs As a result the ciliated epithelium peels off and mixed with degenerated and broken-up polymorphs makes a stringy exudate on the mucosa to form the muco-pus which the patient expectorates This naturally tends to obstruct the bronchial lumen and as air attempts to pass it gives us the stethoscopic sounds of rhonchi and sibili in the larger and smaller branches These sounds may be heard throughout inspiration but if we listen critically we find th y are most insistent in the first third of the breath sound and thereafter tail off towards its end

Fig. 60.
A. Bronchitis.
B. Pulmonary tuberculosis.

This position i inspiration is an important diagnostic f ature as against pulmonary tuberculosis There is no fundamental difference in the quality of the sibili and rhonchi heard in both conditions but the sounds denoting active tuberculosis do not ppear until the third stage of inspiration

and get more and more marked until its end

If the condition is very acute and so produces a hea y viscid exudation this may be seen on the film in larger bronchi as loss of translucency running between thin ill-defined parallel lines The loss of translucency is due to the exudat on th mucosa and in the lumen of the bronchi It is even and homogeneous in type The lines are the congested vessels of the bronchial blood supply which lie in the connective tissu round the walls They are seen running downwards and ut wards t wards both bases in the lowe zones as bronchitis is mainly a basal disease W can t ll they are bronchial shadows as w hall remember from our applied anatomy that as against the blood essels they keep the same width for some distanc and within that distance ha e no branches Usually they re not w ll-defined except near the hilum and they d not continue below the diaphragm as vascular shadows do even with long-continued irritation that is producing th complication of fibrosis As the patient recovers from mpl acute bronchitis these shadows disappear and the film turns to normal There has been no permanent damage t the bronchi the hyperaemia of the bronchial blood supply has not lasted long enough to bring the fixed hanges w find in chronic bronchitis

From their origin it follows that congenital cysts of the bronchi can share in the inflammatory xudation of acut bronchitis This

Sometimes we see d im t increase n th h lar shadow This is due to hyperaemia we hall remember that the h lar shadow are in the main due to blood vessels This increase disappears as the patient reco ers If he has the common type of scoliosis with con exity to the right this reaction usually appe rs more intense and is evident for a longer tim than n the absolutely normal subject in the left hilar region

Chronic Bronchitis

This ondition is especially associated with any prime use of increased blood supply to th lungs this is wh t is so commonly associated with chronic l lar disease of the heart The continual congestion has two ffects on the bronchus —

( ) It contributes to the consid rabl hypertrophy of the mucous membrane The cyl ndric l epitheli m is destroyed and replaced by cubical epithelium so that there is much less viscid sputum than in the acut f rm but on the othe hand in this process the

cilia which should sweep out the *debris* in the lumen, have been lost so that there is much more obstruction by material which is now mainly pus from degenerated polymorphs We see therefore why it is that the abnormal sounds of rhonchi are much coarser than those we heard in acute bronchitis but also why we get from less viscid sputum less loss of translucency within the lines of more inflamed bronchial walls

(*b*) It causes small round cells to invade all the bronchial coats from the mucosa outwards These cells become infiltrated by capillaries and the consequent deposition of fibroblasts with endarteritis of the capillaries leads to increasing fibrosis The main brunt falls on the connective tissue coat because of the long continued hyperaemia of its blood supply

On the film we can recognize it in definite white streaks along the lines of the affected bronchi continuing more clearly and much further outwards from the hilum than any normal striations we may see connected with vascular markings or with the supporting structure of the lung They are particularly evident along the inner bronchial bundles of the lower lobes on both sides only the fact that the heart shadow covers less of the right lower zone makes them more apparent in this area as against the left lowe zone If we stand some distance from th film we can see these streaks give much more evident outlines to the affected bronchi

Fibrosis gives us a new stethoscopic sign beyond the rhonchi of bronchial obstruction We hear dry crepitations throughout inspiration which remain after cough These show us that fibrotic material round the bronchus is resisting its attempt to elongate and open up by the ct of inspiration What was previously only a supporting sur round expanding and contracting with the normal mo ements of the bronchi, is now constricting band As this material is static and not movable as mucus by cough it is a st ady finding in all chronic inflammatory diseases of the chest In its turn it produces emphysema which we shall discuss a little later

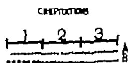


FIG. 61
A. Generalized fibrosis.
B. Marked peribronchial fibrosis.

A still further effect is shown on the film (f the advanced case) in an alteration of the normal cardiac outline due to resulting hyper tension An early sign of this complication appears to be an

increasing size of the aortic knuckle similar to that found in the arteriosclerosis of advancing years Later comes unfolding of the aorta, where this increase of the knuckle is continued downwards and inwards as a band of loss of translucency which joins the bulge of the conus Still later comes flattening of the diaphragm which becomes more and more ineffecti e in its movements from advancing emphysema Experience shows this warrants a grave prognosis indicating that we may shortly see a protuding superior vena cava and the outward displacement of the right auricular border that herald congestive failure

Emphysema

The name emphysema is applied to what is essentially a distension of the alveoli Many types are described in medical literature For all practical purposes in thei immediat and ultimate effects



Fig. 62. Segmental collapse of the right lower lobe in broncho-pneumonia. The line of the collapsing fragment can be seen in the entire lower zone. Outside the defined edge of the affected segment there is emphysema in the costo-phrenic angle.

on lung structures and through this on the patient two types may be distinguished temporary and atrophic

Temporary emphysema occurs with temporary collapse in other words the distension of the emphysematous alveolar walls is complementary and for a considerable time there is apparently as judged by after results no evidence of damage by the stretching of the walls and of the inter lobular connective tissue which return to normal Such a condition is found with temporary blockage of the bronchi small or large W shall see it can occur in localized form in the ephemeral collapse that often complicates broncho-pneumonia (see Fig 62) and in the lobar collapse by foreign body in a main bronchus as in the condition of so-called post-operative pneumonia

The atrophic form is associated with destruction of alveoli breaking down of their walls irreparable stretching up to tearing of their elastic tissue and disintegration of the binding septa which knits them into lobules and links them for support with the peribronchial connective tissue It results from two prime factors first a weakening of the wall that occurs in all chronic illnesses such as bronchitis bronchiectasis and tuberculosis and second a distension from continued cough that destroys alveolar elasticity by the constant stretch of forced inspiration and expiration

There is an intermediate form that occurs in generalized fibrosis, which produces as generalized but slight complementary distension of lobules from shrinkage of their supporting connective tissue. We shall note later how this occurs in pneumonia which heals slowly and badly by repair instead of resolution known as post pneumonic fibrosis the condition commonly affects th whole of one lung Th contracting peribronchial tissue drags on th septa of the lobules bringing a generalized stretching of the elastic tissue of the individual lveoli but it can also become so much thickened that it crushes and destroys the alveoli in its neighbour hood In other words we have both complementary and trophic types

All forms of emphysema occur most easily where the upporting tissue is normally weakest This is why the lung bases so easily blow out in chronic bronchitis why th lower anterior edge of the right lung so readily distends when the middle lobe collapses to fill in the space vacated by the receding l be It is the reason too why with the collapse of one lower lobe th other one so

readily bulges across the anterio mediastinum in an attempt to take its place The lack of support is the cause why we occasionally see localized emphysema over the pices of the lung that may rupture without bvious cause of strain and present us with simpl spontaneous pneumothorax through leakage of air int the pleural cavity (see Fig 12) It is this same natural weakness that will cause the outer and uppe apices t uffer first in blockage of the upper lobe bronchus their lob les collapse ery easily and so lose their residual air Thus t is sometimes poss ble to see slight loss of translucency f even, homogeneous type n the outer infra-clavicular region quit early in case of carcinoma f the upper lobe bronchus

W have already learnt how elastic tissue allows e pans on f th lung, as it responds to muscular stretching throughout th bronchial tree, and how it finally confines th extent of such expansion by its network t rminations round th lveoli N the action takes place when emphysema is present Affected parts have lost their power f mo ement as they are constantly at f ll distension They can be moved only bodily being dragged by other unaffected lung structures Hence the heaving non lat ral movement of the lung bases in chronic bronchitis and the perma nent hyper resonance from constant expansion, whi h has a tympany definitely beyond that of f rced inspiration in normal tissue Prolonged exp rat ry ff rt is to be expected from the sufferer nature s ttempting to f rce air ut of the blown-out vesicles W ee why expiration is now equal in length to o longe than inspiration

Blood vessels in the al eoli and in the upporting connective tissue of lobules are drawn out into thin wiry structures and some are occluded ltogethe with two results First there is icious circle th septa cannot li e without blood supply and so break still furthe second th narrowed essels in th alveolar w lls resist the pulmonary circulation and so reflect on the right he rt and open the way to ll the stages up to congesti failure Close inspection of the film hows corresponding change in th pul monary art ries which are swollen and vident much furth out from the hilum than in the normal film

The reason is not far t seek Such dragging ut and occasional obliteration of th capillaries in the l eola walls means that less blood is open to th residual air there is considerable lessening

of gaseous interchange less chance for the liberation of carbon dioxide and its replacement with oxygen. At the same time there is less movement of the alveoli they cannot get the steady renewal of residual air This is the explanation of the cyanosis of the chronic bronchitic and also of the increasing cyanosis in the bronchial asthmatic He has the added difficulty of the muscular contraction that brings closure of the terminal lumen to the acinus We saw the reason in discussing the muscle fibre structure of the bronchial tree It ends in circular fashion round the last recognizable lumen at the entrance to the acinus (see Fig 21)

The alterations on the X ray film of an advanced case are now easily visualized The rib inter-spaces are widened The vesicles are fewer and larger than normal so there is an increase of translucency by decrease of striations due to their walls The pulmonary vessel markings are more in evidence As there is less venous return the heart is smaller The diaphragm is flattened as the blown-out vesicles press against it We can see the cardiophrenic angles opened out and to percussion we find much decrease in the muscle movement downwards with inspiration The continual flattening slowly brings paresis of the muscle with consequent basal stasis of the accumulated *debris*

CHAPTER XIII

BRONCHIECTASIS

Bronchiectasis means a widening of the bronchial tubes. This may occur over a length of the bronchus in the form called cylindrical or be localized in the form called saccular. It may be congenital in the sense that it is a complication of a prior developmental abnormality of the bronchi, but is usually recognized as being frankly an acquired condition. For its production both obstruction of the lumen and infection of the weakened walls are necessary. Because of the liability of *debris* to accumulate by gravity and stasis in the dependent parts of the lungs we find that most cases are basal, but it may appear in any part of the parenchyma as the result of blockage of its supplying bronchial lumen. Where small branchings are thus involved we have bronchiolectasis. We shall see that this is a continual accompaniment of the gross form involving the main branches of the lower lobes.

We now recognize that bronchiectasis is not of necessity a lobar disease. Certain segments are known to be particularly affected. These are the basal segments of the lower lobes, the left more than the right, the lingula of the left upper lobe alone or with lower lobe basal segments, and the right middle lobe. If other single segments are diseased they are almost invariably the end results of previous infections. We would therefore suspect that bronchiectasis confined to the superior segment of the left lower lobe had resulted from a lung abscess in that segment.

It follows that proper treatment will depend on proper diagnosis, which means full investigation of the whole bronchial tree. Lipiodol is therefore injected nowadays under screen observation so that the operator can ensure filling of every broncho-pulmonary segment.

The pathogenesis is most easily understood if it be envisaged as a further development of chronic bronchitis which has become ulcerative. After weakening the wall by its chronicity it has destroyed all the components of the wall from the epithelium to the muscle and elastic fibres and has finally penetrated the cartilage to infect the surrounding parenchyma.

upper sinus infection This is a ery common finding often accompanying an acute antrum trouble Expert opinion on the upper sinuses including X ray should of course be obtained but the following points may aid in comparing the chest films

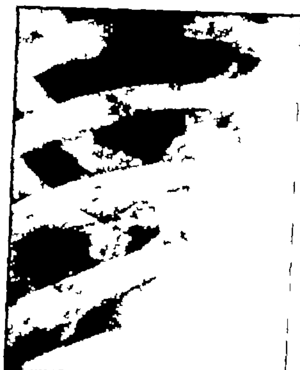


Fig. 65 The right cardio-phrenic angle in upper sinus infection.

The film due to upper sinusitis usually shows much more homogeneity in its basal shadow we do not get the same evidence of fibrosis unless the condition is of ery long standing with continued reinfection of the bronchi when of course the patient is developing resultant bronchiectasis Furth r the film shows a confused shadow of the inner third of the diaph agm merging into that in the cardio-phrenic angle W do not see the lower bronchi curving inwards We re looking at hyperaemia of vessels continuing below th diaphragm

If we look at the film of bronchiectasis at some distance from the viewing box we shall see that the striations stand out clearly as

well-defined parallel lines here and there interrupted where the cartilage is disappearing. Around them we see small opaque and hard looking shadows which are due to the collapsed and organized lobules well defined because they are offset by others among them which have complementary emphysema. That is, the disease is extending by bronchiolectasis.

The next stage is perforation of the bronchial wall and consequent extension of the sepsis into recognizable areas of parenchyma. Its occurrence is heralded by the stethoscope finding of râles which mean that parenchymatous material is entering the bronchi. If we listen critically we shall find they are in the middle third of the inspiration, that is the bronchial parenchymatous phase. They rise in intensity to it from the first phase and then fall off in the third. They are due to air entering bronchioles into which catarrhal exudate is discharging from lobules that have been filled with that first inflammatory fluid which is the indication of irritation of the walls. In other words they are the râles of localized lung abscess.

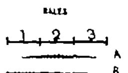


FIG. 66.
A. Lung abscess.
B. Bronchiectasis with lung abscess.

We can recognize this exudate on the film. It is the "oedema" of many radiological reports and is always homogeneous in its shadow, having a light loss of translucency comparable with that thrown by the inferior vena cava. In this particular instance it is seen on the outer edges of a more opaque and likewise homogeneous shadow cast by lobules that have passed the stage of inflammatory exudation and are now filled with septic material. We know it connotes advancing bronchiectasis only because we read with it the characteristic shadows of fibrosis and organization detailed above. It appears with each exacerbation of the disease, later gets denser as becoming true septic exudate and later still shows all the non-homogeneous changes of collapse and fibrosis.

What we have now is a cavity in the bronchus connecting with a cavity in the lung, septic bronchi are continuous with septic parenchyma. To all intents and purposes we have in several areas what we shall find later is the end result of chronic lung abscess, and where the septic lung has infected the bronchus supplying the diseased broncho-pulmonary segment. We shall find that the râles have taken on distinct metallic consonating quality. These sounds are now present throughout two-thirds

of the inspiration over a considerable area of the lung field Lipiodol introduced at this stage will demonstrate every step of the destructive process here we shall see only cylindrical

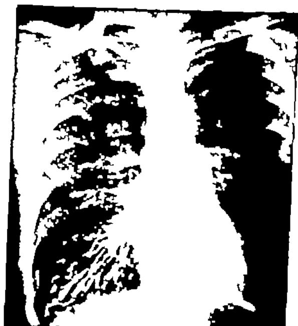


FIG. 67 Lipiodol in basal bronchiectasis.

enlargement there sacculation, at still another point nothing resembling a bronchus but only a large irregular opaque area with cotton wool edges which merge with the finer loss of translucency that indicates catarrh in irritated lobules

Mediastinal shift will be more evident still to both film and physical findings The fingers will show gross clubbing of the drumstick septic type Dullness will be much more marked and the foul sputum will contain the elastic fibres of the destroyed parenchyma.

Bronchiolectasis

We have noted how bronchiectasis multiplies itself in the minor form of bronchiolectasis by the involvement of bronchioles. Such a condition is almost always a secondary one the lumen has been blocked and then infected by septic material in the lobules

it supplies a process which copies the pathogenesis of adult tuberculosis W shall see in the next chapter that its common cause is primary broncho-pneumonia which has not been abl to get rid of its exudate It is always a fai ly localized condition and this helps us to differentiate it as picture that has lements not dissimilar to those of broncho-pneumonic tuberculosis where the process is much more widespread into the mid zones

The condition is not the same pathologically or radiologically as bronchiolitis condition which is discussed in the chapter on Miliary Shadows

CHAPTER XIV

THE PNEUMONIAS

THE pathogeneses of pneumonia and *broncho-pneumonia* are fundamentally the same consisting in the interstitial reaction to the infecting organisms In both diseases the organisms penetrate the wall of the bronchus to its surrounding connective tissue and by its lymphatics are carried to the walls of the alveoli which they first irritate, and later penetrate In this way the pneumococci of pneumonia and the streptococci of broncho-pneumonia *differ from the bacilli of influenza* and *Friedlander s bacilli* which we have noted are carried direct to the lobules by th i supply ing terminal bronchioles

The connective tissue puts up a poor resistance to the pneumococcus In the great majority of cases th organism seems to penetrate the wall of a main bronchus such as that to the right lower lobe close to its origin It finds no protective barrier against its invasion of the parenchyma and therefore rapidly involves th whole lobe On the other hand the connective tissue puts up an intense resistance shown in marked inflammatory reaction to the streptococcus so that it confines the disease to patches of lobules We can see why loba pneumonia is a disease which spreads from the hilum in radial fashion Its progress is rapid the invading army is too strong and sweeps aside any light opposition it may meet It is possible however now and again to see an example where pneumonic exudate is already dev loped near the hilum before the first evidence f inflammatory reaction is evident in lobules nearer the periphery

The manner of onset explains to us also the manner of clearance by ither resolution or repair which must be again radial from the hilum to the periphery The bronchi ha e a chance to recov r from any slight reaction in their connective tissue before they are called on to deal with the parenchymal exudate which will flood them in due course to produce the typical pneumonic sputum This manner of clearance of th disease is an important one in differential diagnosis by film examination It is the opposit to that seen in the so-called "epituberculosis of childhood

Serial fil s will help us to decide which w are d aling with. The picture of pneumonia le rs rapidly afte the crisis but epituberculosis will tak many weeks n months to resolv and w ll lea on the film enlarged and crenated hilar glands and perhaps lso w ll marked Ghon s focus at the sit of the original infection t wards the periphery of the ffect d part

Let us try to f llow the pathogenesis f dev loping pneumonia in more det ll as w see it on the film If there is any reaction n the onnect tissue t the penetr tion of the bronchial w ll by the pneumococcus w se ncreased triations along the lines of bronchial branching due to congestion by polymorphs in the peribronchial peri ascula and nt lobula onnecti e tissues These may be evident the out r zone of the ffected lobe while t inne re gi es a homogeneous loss of translucency This hadow s f the same type as w found in the tension of sepsis from bronchus to parench ma n ad anc ng bronchiectasis On the out edge we see the same fine loss of translucency due t the non-specific irrit tion of al eolar w ll N rer th h lum w have the dense hadow of true pneumonic udate onsisting of polymorphs some large mononucle rs and lymphocytes, n serum ont ining rying quantity of fibri dependent on the onnecti tissue reaction The lobules so affected fill quickly and distend and this tret hing of the r lastic t ssue h lps them t ont act lat r so that the an e uat the udat nt the r bronchioles

It is fundamental t the understanding of the physiologic l effects the X ray film and the ph ical igns, to realize that when pneumonia is nc mplicat d this udat eplaces the residual r l es rs sense The same phy iologic l action as that of residual r remains on the lastic tissue of the bronchi The alveol re filled t f ll apacit and the lastic netw rk of the l e l is theref re n tret h just as it would be in f ll inspiration There is no blockage of t rminal bronchioles, nd therel re no ollapse of lobules and no resultant hift of the mediastinum to the id of the disease The same thing pplies to uncomplic ted broncho-pneumonia and uncomplic ted lung absc ss so that they are tot ll unl k in pathogenesis physiologic l ffects and physic l igns to both bronchiectasis and adult phthisis In these latt r are prolif rativ diseases The use rganization and llapse of lobules and has from thei beginning associated fibrosis They will therefore ause the mediastinum to shift tow rd the part of

the lung they affect so that we shall find the heart and the trachea displaced towards them

Once fully developed pneumonia outlines the lobe anatomically and the dense homogeneous shadow of its exudate remains until the crisis This being so the shape and density in any one lobe must vary with the shape and alter with the depth of tissue in the normal lobe as we saw in our reminders on applied anatomy The right middle lobe must be defined and dense just below the line of the lesser fissure and become more translucent towards the lower zone and unless the lower lobe is also invol ed we must see a clear area between the lower limits of loss of translucency and the diaphragm because this area is occupied by the lower lobe (see Fig 25)

In the same way the shadow of lower lobe pneumonia, densest in the lower zone over the diaphragm must lessen steadily upwards *towards the mid zone It is sometimes confused with that of* tuberculous pleural effusion The physiological effects to be expected in these conditions should aid us effusion pushes the mediastinum to the opposite side where we shall find a positive sterno-mastoid sign If we look critically at the shadow of tuberculous pleural eff*usion we shall see that its peripheral density is far* less than that of pneumonia and that w can usually see the ribs through it (see Figs 5 and 1 6) Its loss of translucency increases steadily towards the middle of the lung field giving an impression of a band running from the axilla downwards and inwards to the diaphragm The effusion is compressing the lung and we are looking at the area so compressed when we see this band of greater density This is quite unlike the pneumonic density which is equal at any one level throughout its extent along a *horizontal line* drawn between the periphery and the mediastinum Here the density must lessen as it goes upwards as the lobe thins out to *its* lingula in other words there is less and less depth of parenchyma to be filled by the pneumonic exudate

Moreover we shall remember the further aid to differential diagnosis An uncomplicated pleural effusion will not blot out the vessel markings in the underlying lung but exaggerate *them* as there is hyperaemia but pneumonia blots out *all normal striations* and is usually so dense in its shadows that we cannot make out the *outlines of the ribs*

The picture of fluid complicating lower lobe pneumonia can now be readily visualized for it combines both pictures Towards

the periphery we shall see the lighter loss of translucency of the fluid and through it we can generally see th ribs quite clearly as far as the outer edg of the denser shadow cast by the pneumonia No lung markings will appear in either shadow Now the media stinum will be to the opposite ide whereas it was entral as long as the primary pneumonia was uncomplicated Before the fluid developed there was no sterno-mastoid sign now the muscle on the opposite side is under distinct tension

From what w ha e learnt so far it follows that the slight and temporary shift sufficient to be noted by film examination in some cases of pneumonia is due either to overdistension l the lobules by thei exudate or t an ephemeral blockage of their bronchioles Close inspection of the picture should t ll us which w are lewing for the ffect of the former will be traction on the mediastinum without evidenc of ollapse the latter will present accompanying evidence of what has taken place For exampl if this has occurred as omplication of right upper lobe pneumonia we hall see a distinct lowe edge of the density in a sharp line This is proof that the lingula of the lobe has shrunk upwards to the line of the lesser fissure whi h becomes convex upwards and is higher than its thin hai like streak we can see about the third or fourth int rspace on the normal film Furthermore the uncomplicated pneumonia showed normal blood vessel markings in the middl and lower lobes but now these are splay d out in order to supply the emphysematous mid and low r lobes

To physical igns pneumonia gives us dullness due to exudat A the lobular lastic tissue is on full stretch and maintains the inspiratory position of the bronchial walls w have no movement of the affected part There w ll therefore be bsence of true breath sounds but bronchial breathing will occur as the air ent ring the mai lobar bronchus at its origin is conducted through the consolidation which acts as sounding board

The method of learance of the pneumonias and the rapidity of the process is a fortunate ffai for the patient Considering all the possibilities f complication w may well marv l that w see so few cases f resulting fibrosis f the peribronchial and peri vascular tissues so ecently recovered from thei nitial reaction to the invading organisms ha e now to deal with lymphocytes and plasma lls These again produce triations on the film and they can remain f a considerable time They should not lead us to giv bad prognosis by reading them as evidence

fibrosis unless e find they are accompanied by the characteristic crepitations of this condition But they can produce fibrosis as we shall discuss in more detail later

The *fibrinous exudate in the lobules* is coughed up or absorbed after being liquefied by a proteolytic ferment supplied by its accompanying polymorphs It is because of this *liquefaction that we* hear the so-called redux crepitations of pneumonia If we listen to them critically we shall find that they are really sticky sounds at the ry end of inspiration and are râles rather than crepitations We are hearing air entering the al eoli through the exudate which is no discharging i to terminal bronchioles

It may be that poo supply of polymorphs in the exudate is the basic cause in those cases that go on to fibrosis capillaries ha e time to enter it before it is liquefied In any case we do know that fibroblasts replace the fibrin passing through the alveolar walls and from lobule to lobule causing organization of the e udate that remains and consequent shrinkage collapse Although others around these lobules are bound to show some complementary emphysema *there will be shift of the mediastinum to the affected* s de normal unaffected tissue is being dragged o er by the intr pleural pressure Chronic int rstitial pneumonia a it is called is therefore exaggerated repair tissue It is usually found in one lung and we can see how it differs from the fibrosis of chronic bronchitis due to bilateral long-standing hyperaemia because this latte is a peri bronchial tissue increase not an i ter lobular one If it is very severe it can crush the supplying bronchi les and gi e us scattered areas of lobular collapse which is not an unusual finding

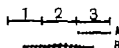


Fig. 64.
A Pneumonia.
B Broncho-pneumonia.

It follows that the X ray of chronic interstitial pneumonia will show the ribs on the ffected side clos r together than normal and that the diaphragm will be higher than normal while the heart and mediasti um will hav drifted towards th fibrosed lung Throughout the diseased lung ill be trands of fibrosis which have no distribution corresponding with that of the *normal blood supply* These ill be more prominent n distant iewing and will be seen to ha e *scattered among them nod lar like shado s* which are cast by organized bunches of alv l

Auscultation will gi us widespread repitations hich reach their loudest point tow rds the e d of inspiration as they are parenchymatous in origin

This condition is quit unlike the results of the pneumonia of the new born because here at lectasis is preceding entity the lobules ne er opened at ll which is necessarily a d fferent condition from acquired collapse It is also quit unlik so-called post-operati pneumonia which is not pneumonia, but a purely bronchial disease A udden thick sticky exudat from the wall blocks the lumen with the same dramatic effect seen with an inhaled foreign body Residual r cannot be renewed the parenchyma collapses and there is no obstacle to the action of the intra pleural pressure on a lobe that perforce must hrink to its main bronchus at the h lum Screening the patient or taking two films one on complete insp ration and the other on omplet expiration will show us what neve happens in ordinary pneumococcal pneumonia wing of th collapsed lobe the heart and the mediastinum with ach inspiration to the affected ide There will be some ecov ry w th ach xpiration but still displaced mediastinum which w ll be shown by the posit terno-mastoid sign even t be mpossible to d monstrate by the position of the apex beat If the glutinous mat rial i not cle ed out by postural drainage or bronchoscopic suction the consequences can be ery serious Organization can lead to every stage of bronchiectasis the same end esult so often seen the pneumonia of the new born To this last ondition the name ongenital bronchiectasis may be applied with its at lectasis present from birth there is often mal-dev lopment bronchi so that cysts are frequently found and these the supe mposed infection turns into umps of septic mat rial to add to the bronchiectatic end result

From what has been said t the pathogenesis of pneumonia the possibility of localized forms can be asily en saged The orga nisms tra el some way along the main bronchus bef re piercing the w ll of one branch This is omparati ly ommon in the pneumonia of childhood which gives e of its forms the so-called hilar pneumonia by n hing the posterio horizont l branch of the right lower lobe bronchus It is not uncommon to find in a adult that only the pectoral or the axillary branch of the right upper lobe bronchus has been affected so that w see on the film that triangula hadow of the ne f parenchyma alr ady referred to in the section in applied natomy (see Figs 14 and 5) The dev lopment of the disease is just the same as in lobar pneumonia It spreads outw rds from the point at which the

bacilli penetrated the bronchial wall so that all the segment of lung supplied by that bronchus is involved

The shadow cast by these localized pneumonias is never really like that of tuberculosis with which it is usually considered in differential diagnosis Both may give a triangular loss of translucency in the outer axillary region of the right upper lobe the pneumonic one being due to involvement of the axillary branch of the main bronchus while the tuberculous one is due to a tuberculous deposit in the periphery of the outer base of the upper lobe that is commonly just above the peripheral end of the lesser fissure Both cause thickening of the fissure Both can show abnormal films for a long time after onset because localized pneumonia tends to heal by repair rather than by resolution If however we look critically at serial films we shall find that the tuberculous shadow always has its main continuing density against the periphery as it is essentially a parenchymatous disease the bronchi are involved only secondarily at all stages This is not so



FIG. 69 Recovering partial pneumonia of right upper lobe

with the localized pneumonia healing by repair the main density is now at the apex of the triangle that is round the supplying bronchus at its ntranc Secondary invol ement of bronchi in tubercle is at the ite of the deposit; there the bronchi dilate steadily until, in most cases definite cavitation is seen

Another shadow that may take considerable time to clear after pneumonia is that of the exudate on the pleura especially if the interlobar pleura has been affected In all cases there is a true

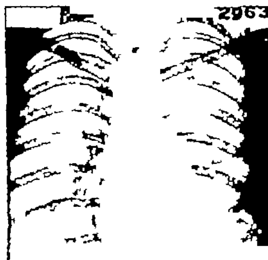


Fig. 70. Filling of left costo-phrenic angle in recovering pneumonia.

pneumonic exudation on the visceral layer and this causes adhesion to the inflamed parietal layer mo vement by inspiration therefore causes sharp stabbing pain Liquefaction usuall leads to rapid absorption but w ha lready noted how absorption may take long t me in the costo-phrenic angle and in the i t rlobar fissure so that w get i lling of the costo-phrenic angles and tenting of the diaphragm that are permanent but of no cl nical import In the

same way the lesser fissur may be thickened for a considerable period after a partial pneumonia



FIG. 71 Lateral view of fluid in main fissure producing "banding."

Broncho-pneumonia

Broncho-pneumonia as already stated may be considered as consisting of localized patches of pneumonia confined by the strong resistance of the peribronchial connecti e tissue which limits the disease to bunches of lobules It is commonly bilateral from its onset, but it may be completely uni lateral, o develop on one side later than the other It is usually in the lower lobes but is not uncommon in the right middle lobe

Its progress can be followed on the post ro-anterior film, which demonstrates shadow that begin near the cardiac bord r and increase in size and in spread along the bronchial distribution so that they go downwards and outwa ds in lower lobe involvement and almost horizontally outwards in middl lobe disease Th in olved areas show th same homogeneous losses of trans-lucency as we saw in pneumonia only now they are in thei greate densities triangular in shape

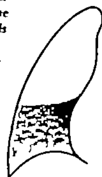


FIG. 72. Broncho-pneumonia, right middle lobe.

particularly in the middle lobe as the lobules are pictured along the sid of their cone-shaped broncho-pulmonary segments The



FIG. 73. "Contact" film of right lower lobe in Fig. 54

lobules inv l ed by pneumonic exudat are nev r entirely discrete because they ha round them those showing the first inflammatory non-specific catarrhal udate Ther is no hift of the mediastinum.

The picture is therefore in two cha acteristic unlik that of broncho-pneumonic phthisis Firstly this disease is fibro-caseous and w ll show le ,cut areas of acinar collapse among others with omplement ry emphysema spite of the fact that both conditions ma ha surrounding oedematous hadow This w ll be quit evident if w look t the films at some distanc from the iewing bo when the shadow of streptococcal b oncho-pneumonia will be no more ident but those of tuberc lous broncho pneumonia will become much mor clearl t Se ondl there w ll lw y be some llapse and ther f re some displa ment of the mediastinum w th phthisis and il this not k to X ra t w ll be to ph sk l exam nation

The ph sc l find ngs n be explained by the pathogenesis and the X r y pictu W hall find pat hy reas of lene os lobules filled t distension with pneumonic exudat and immed iat l the disease is f ll des loped w hall get gra of this exudat ent ring the t rminal bronchioles The lobe n lved is never

of course completely silent there are parts which have escaped altogether and others where only inflammatory exudate occupies lobules still maintaining some airway If we listen carefully we hear rather bubbly râles in the mid third of inspiration because this exudate is discharging into terminal bronchioles (see Fig 68) This is a distinctiv stethoscopic finding throughout the clinical illness the only difference being that the râles get more marked as the pneumonic exudate liquefies

The dullness will naturally be much less than that of pneumonia as the involvement is patchy but there will be distinct lack of movement laterally in cases where the lower lobes are diseased

By reason of the much more intense connective tissue reaction many cases proceed to small areas of atelectasis The increased tissue naturally gathers round the terminal bronchioles of the affected lobules This interferes with the natural movements of the bronchioles in response to respiratory action; that is their muscle fibre action is inhibited Their elastic tissue reaction which is dependent on the stretching of the alveoli to residual air is already interfered with, since the alveoli are fixed at full expansion by the exudate in them When the disease tries to resolve these bronchioles are therefore caught at a distinct disadvantage. They have no time to recover from this initial irritation of their surrounding connective tissue before they are flooded by lymphocytes and plasma cells from the exudate now trying to escape from the alveoli as resolution sets in We have now irritation of the inner wall of the bronchioles added to their connective tissue irritation on their outer walls They are therefore all too easily blocked so that alveoli that have managed to empty go on to collapse while those still containing exudate go on to rganization

We can see therefore the inherent danger of too arly movement, perhaps advised by the belief that modern methods of treatment by reducing temperature ha e brought resolution If this is dangerous in lobar pneumonia it is much more dangerous in broncho-pneumonia If the bronchioles do not empty their walls weaken and the way is open to bronchiolectasis and later to bronchiectasis

This is especially possible in right middle lobe disease We have already noted that its main bronchus goes out almost horizontally from the right main bronchus It is therefore difficult to drain If it goes on to blockage in its terminal bronchioles we have a slowly changing picture and accompanying changes in

physical igns The areas that on previous distant viewing merged with thei surrounding catarrhal inflammatory exudate now stand out with the larity of broncho-pneumon c phthisis with which we may confuse them because of the accompanying shift of the mediastinum W shall not howev r that they are confined to lateral dens ties i one area of the lower mid zone as against the widespread distribution of t berculous broncho-pneumonia and that they hav lw y much more exaggerated peribronchial fibrosis A lat ral film w uld show us how much more the lesser fissure shares in this fibrosis than t does in phthisis and of course, the general cond tion and th lower and less-sw nging temperature will be a f rther d Both ond tions will f course give us the crepitations of fibrosis but they w ll be much more localized and definitely coarse and more insistent in c mplic ted streptococcal broncho-pneumonia. Many cases seem to go on to impl fibrosis in the lower mid zone Indeed the fortunat patient may escape further clinical upset but many go on to lowly increasing bronchiolectasis and t established and progressiv bronchiectasis This is a common end-result long aft rwards by superimposed infections

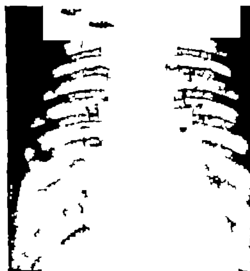


Fig. 74 Fibrosis following right middle lobe broncho-pneumonia.

The patient may for a long time be unaware of what has happened. Mass radiography has shown that many apparently completely



F 75 Lordotic view of same patient as FIG. 74

recovered cases of middle lobe broncho pneumonia have been left with much fib os s The X rays sho n in Figs 74 and 75 are an example The first demonstrates a loss of transluc ncy in the right lower mid zone The patient had signs of fibrosis in this area The lordotic picture shows how the middle lobe has shrunk ery considerably Lipiodol later pro ed the presence of bronchiectasis

An mportant point to remember is that e ery case of bronchopneumonia causes marked increase in Hila shadow because of the intense hyperaemia of its resistance the hilar glands also swell with oedema This hilar i crease takes a long time t clear up and this is especially noted ith the usual type of scoliosis which brings the left hilum into prominence There is the possibility of gra e diagnosis of localized glandular disease but the swelling is seld m of the extent of Hodgkin s disease and the swing of the P l Ebstein feve and the blood count will be further aids in doubtful cases The enlargement remains long after the patient is linically well

CHAPTER XV

ATELECTASIS

In dealing with this condition wh ch may be defined as collapse f parenchyma through loss f residual air w shall confine ou discussion t the acquired f rm due t obstruction of the lumen supplying a broncho-pulmonary area This may occur by some thing inside the bronchus such as a growth o inhaled foreign body blood mucus o granulation tissue or by something pressing on the wall of the bronchus such as an enlarged gland or tumo formation As we saw in our studies of applied anatomy and in the description of post broncho-pneumonic atelectasis in the right iddle lobe the size of the affected cone depends n th size of the bronchial lumen Th same w shall find to be true in the secon dary results of blockage of th terminal respiratory bronchiole in ad It phthisis where gran lation tissue enters it from the primary acina deposit If main bronchus is occluded, as in post operative pneumonia then the whole lobe collapses since the residual ai to every t rminal alveolus is absorbed

In such condition oxygen is first absorbed by the capillaries in the alveolar walls and w get on the film fine loss of trans lucency W cannot see this taking place in the small telectatic reas of bronchiectasis o complicated broncho-pneumonia but w can follow it quit asily where a whole lobe is shutting down, and we shall lw y find thi light homogeneous shadow beg nning in the least supported areas of parenchyma Thus in the upper lobe it w ll occu first in the outer apex and in the lower lobe along the anterio basal margin; we saw the reason in discussing tructural upport under our onsiderations of appl ed anatomy these reas are dependent almost complet ly for their normal position on the esidual ai and ha but little support from thei scaffolding f onnecti tissue Later the nitrogen is bsorbed and the shadow while till homogeneous becomes definit ly more dense

Up to the time when such density covers the whole of the affected lobe it may be possible in those cases that are due to carcinoma of the bronchus to d fferentiat on the film the he y opacity of

The patient may for a long time be unaware of what has happened. Mass radiography has shown that many apparently completely



F G. 75 Lordotic iew of same patient as Fig. 74

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Up t the time when such density cov rs the whole f the ffected lobe it may be possible in those cases that are due to carcinoma of the bronchus to d fferentiat on the film the heavy opacity of

the actual growth near the hilum, next to it the slightly less opacity of those areas already *completely collapsed and towards the peri* phery the finer loss of translucency of early collapse We have here a characteristic whitewash brush film It looks as if the brush had been applied heavily over the point of entry of the bronchus and then as it was swept outwards towards the periphery had been pressed with continuing less force The apex of the broncho-pulmonary area is densest its base least dense Later as the lobe becomes smaller by shrinkage and is followed up by its proximal lobe which develops *pari passu* its complementary emphysema the interlobar fissure rises and the density becomes throughout equal to that of the causative growth, which indeed in most cases has by this time extended by its cells, or its mucinous exudate into a considerable portion of the surrounding parenchyma

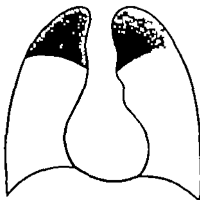


FIG. 74. Carcinoma left upper lobe bronchus, compared with tuberculous pneumonia of right upper lobe.

In many there appears by now the further complication of abscess bringing a more or less central area of translucency

From the beginning there will be lack of movement and dullness will deepen with increasing involvement of the growth. The mediastinum will drift to the same side We shall find the evid ence of shrinkage in alteration of the normal lesser fissure or the appearance of the greater fissure on the postero-*anterior film* and in the change in blood supply striations in those parts of the lung that are blown out by complementary emphysema Lateral films are of particular assistance as we have already noted *in all conditions* effecting change in size and shape of lobes

It follows that the dullness of collapse must be accompanied by the tympany of emphysema This is why broncho-pneumonia and its complication of terminal bronchiole blockage never give over all dullness and why with the dead dullness of the collapsed right upper lobe we find marked tympany in the compensatory distension of the middle and lower lobes

Bronchial breathing may be very marked just short of the actual growth at the origin of the affected bronchus. Added sounds inside the collapsed lobe do not present invariably in the



Fig. 77. Carcinoma right upper lobe.

upper lobes unless there is damming back of secretion and the complication of abscess is superimposed. Once the extension of growth begins to press on the bronchus to a lower lobe we get, beyond this second bronchus, a very high pitched musical sibilus from air passing the obstruction, as in the production of an instrumental whistle. Its appearance is of grave import.

The most constant sign of collapse in all its forms is the sterno-mastoid sign, which is always positive on the affected side. In the difficulties of differential diagnosis it is a much more reliable finding than any other single aid, even including comparison of films on complete inspiration and complete expiration. We have already noted how the trachea reacts to changes in normal lung elasticity, the shrinkage of the affected part leaving the remaining normal tissue open to both increased pull on the same side and increased push from the other. While this tracheal reaction is

constant the position of the heart is variable for as we saw its greatest weight is in the lower mediastinum, and it will react only to lower zone disease or complete unilateral disease Even then it is unwilling to respond to anything min r in force It will swing on its long axis and can be pushed backwards by the emphy sema of the opposite side before it has moved bodily and laterally to the affected side While it must certainly always be located by palpation and by stethoscope the position of the apex beat in collapse may be a misleading sign E en to screen examination the heart may show no definite shift with inspiration if the diseased area is above the origin of the aorta These tests we shall see *applied in more detail in considering atelectasis of individual lobes*

Upper Lobe Atelectasis

The commonest cause is carcinoma of the bronchus *It is here* we see best demonstrated the whitewash brush effect on the film although the very first evidence of interference with residual air may appear in a fine loss of translucency in the outer *infra* clavicular region. Quite early we can see the accessory signs in shift of the trachea and the mediastinum to the affected side and in the upward shrinkage of the interlobar fissure on to which the thin triangular lingula retreats steadily The interlobar fissure becomes more and more evident because the rays are passing through more and more tissue It has added to it all the tissue of the retracting lingula of the lobe These findings are always more easily ecognized on the right as against the left side as the shadow of the upper right cardiac border is made in great part by the movable superior vena cava, usually less w ll confined than other *mediastinal structures by the surrounding areolar tissue* The heart does not mo e much in the lower two-thirds of its extent and so th re may be no appreciable change in the position of the apex beat to palpation or auscultation even *with considerable* shrinkage of the upper lobe

It is quite possible that in its early tages the film can resemble that of a dev loping pneumococcal pneumonia *although tempera* ture symptoms and the examination of serial films should help us to avoid misinterpretation, and we know that an *uncomplicated* pneumonia does not produce mediastinal shift *A lateral film* will aid us because we shall see the retraction of the lobe on to the inner end of the lesser fissure in a manner nev r displayed with developed pneumonia Further we shall find that while pneumonia gi es

dullness both t n rly and posteriorly that of collapse is alw ys anterio and belo the inne end f the colla bone t the sit of rigin of th bronchus

Th diff renc in s t f dullness is f distinct help if the diagnosis is against upper lobe pneumonic phthisis h re the dullness is posterio as tubercl in adult f rm is post rio disease inclined to ttack parenchyma supplied by d rsally inclined branches of the b onchial tree Again tuberculosis is parenchymatous disease in its pathogenesis not bronchial disease and the greatest density in its pictu is noted below th middle third of the collar bone and not the t f the entranc f th ain bronchus

Right Middle Lobe Atelectasis

W no ed when dealing w th right middl lobe bronchopneumonia that the dangers of inefficient bronchial blockage are considerabl wing to th angle t wh h the main loba bronchus

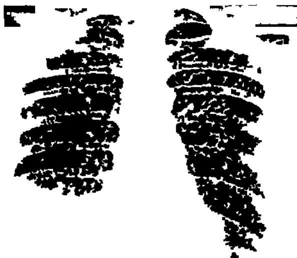


FIG. 78 Collapsed right mid-lobes from broncho-pneumonia.

comes off the right bifurcation Post pneumonic llapse by ucid sputum is ery common and occ rs n many cases with perhaps little immediat but grave afte -effects It can come on quit

suddenly or very slowly In the latter case the pain has a characteristic position along a band below the right nipple coming in waves of intensity up to a pitch of severe discomfort

The postero-anterior film gives a shadow outlining the lobe, heaviest along the line of the lesser fissure and lessening to an area of translucency above the diaphragm in spite of the fact that

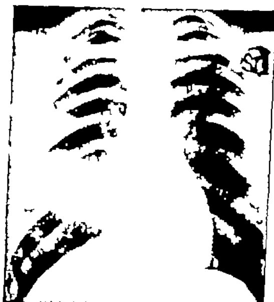


FIG. 79 Collapse right middle lobe, by innocent intra-bronchial growth.

this muscle rises towards its position of expiration against the emphysema of the lower lobe The lateral film is characteristic and unlike that of any other disease It is almost always of the whitewash brush type dense at the entrance of the bronchus and fading towards its base The upper side of the triangle is along a line beginning at the junction of the middle and lower thirds of the mediastinum and going horizontally outwards to the posterior aspect of the sternum The inner side of the triangle goes downwards and outwards to a point near the middle of the diaphragm The case demonstrated in Fig 79 was one of collapse due to

benign tumour of the middle lobe bronchus the growth was removed through a bronchoscope



FIG. 80 Lateral view same case as in Fig. 79

The lateral film of collapsed middle lobe can cause difficulty in diagnosis as against effusion in the lesser fissure The upper



FIG. 81 Lateral view in interlobar effusion

border of th fluid is seldom straight as in collapse It is inclined to bulge up ards in con ex fashion and e en if it is convex do n wards because the fluid is no v en ysted v e do not get evidence of emphysema in other parts of the lung Physical signs should also help The fluid will be encysted dense and restricted in area before it causes drift of the mediastinum to ards it and it will not give us the marked tympany of complementary emphysema which is bound to accompany collapse

The swing of the heart to the affected ide by inspiratio is more marked in middle lobe than that found in upper lobe collapse The two characteristics to physical examination are the positi e sterno-mastoid sign and dullness which i anterior in the lower mid zone Th emphysematous lower lobe gives definit posterior tympany

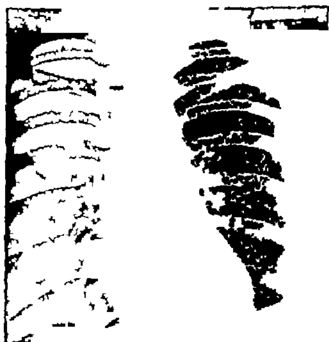


FIG. 82. Fluid in left interlobar fissure.

The only oth r condition giving uch marked a terior dullness is fluid in th main interl bar fissure but its films antero-posterior lordotic and lateral are entirely diff rent the first showing a low

of translucency sitting bo e the diaphragm lik that f pleurisy secondary t sub-phrenic abscess, the lordotic tent lik shadow with its base o e the diaphragm and the lat ral density between the cardiac shadow and the diaphragm

As already noted many cases of post broncho-pneumonic collapse of the middle lobe can pass unrecognized t the time of the clinical illness as there is much less upset in pulse and temperatur than occurs in the average case of lower lobe post-pneumonic collapse Only considerably later aft r what then appears to ha e been a rather protracted convalescence does the patient complain f dragging pain; full investigation including lipiodol injection, commonly shows consid rable d latation of the bronch Bronchi ectasis is by no means rare sometimes by super imposed infection but often only by the continuing effects of the collapse which has led to bscess formation by the damming back of the exudat

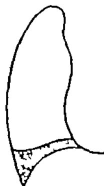


Fig 83 Pleurisy secondary to abdominal disease.

This type of case often presents one of the most difficult problems f differential diagnosis because the abscess lmost alw y produces the complication of a frank pleurisy with the danger of consequent empy ma We are now presented with a condition which is all too easily read as pleural effusion Fortunately th tracheal displacement in response to t lectasis is alw y more marked than the pressure of the fluid n the w rds th sternomastoid sign remains positiv on the right The writer has seen several cases where this was the main indication to the true diagnosis It is of ourse taken for granted that all othe linical aids including needling w ll be employed for every ultimat diagnosis

Lower Lobe Atelectasis

The commonest types result from pneumonia broncho-pneumonia post operative pneumonia nd new growth of th bronchus Of these the most udden in onset and th most massive in result is the post-operative du as w hav lready noted to the formation of a glutinous udat on the wall of the main bronchus The pneumonic and broncho-pneumonic types

border of the fluid i seldom straight as in collapse It is inclined to bulge upwards n convex fashion and e en if it is convex downwards because the fluid is now encysted we do not get evidence of emphysema in other parts of the lung Physical signs should also help The fluid will be encysted dense and restricted in area before it causes drift of the mediastinum tov ards it and it will not give us the marked tympany of complementary emphysema which is bound to accompany collapse

The swing of the heart to the affected sid by inspiration is more marked in middl lobe than that found in upper lobe collapse The two characteristics to physical examination are the positive sterno-mastoid sign and dullness which is anterior in the lower mid zone The emphysematous lower lobe gi es definite posterior tympany



FIG. 82. Fluid in left interlobar fissure.

The only other condition gi ing such marked anterior dullness is fluid in th main interlobar fissure but it films antero-posterior lordotic and lat ral are entirely *different* the first showing a loss

this has less structural support, as well necessarily as less residual air for absorption The blood essels of th partially collapsed lobe are crowded towards the mediastinum which is displaced to the side of the lesion With this the most marked change in physical signs is the sharp tension of the sterno-mastoid o th same sid It was unaffected by uncomplicated pneumonia At the same time w note that the d llness which had been learing both anteriorly and posteriorly is now again marked posterio ly this is because the lung shrinks downwards and backwards as well as inwards The last two mo ements tend to make the heart swing on its axis: thus in right lower lobe collapse th e right low r border recedes and th left lower border comes forwards and to the right. The apex be t may therefore be but littl disturbed from the normal, and is not a reliabl aid to the diagnosis The swing of the heart on inspiration to the affected side will be quit evident to screen examination

In the second stage the shadow is much more dense its o t border goes downwards and outwards towards the mid-point of the diaphragm on the affected sid So dense is the shadow that it appears t be continuous with and equal to the heavi est density of the heart shadow towards its mid-point. There is therefore total oblite ation of the cardio-phrenic angle It follows that this shadow ought not to be confused with that of squat heart in cases f left lower lobe collapse True the diaphragm is higher than normal in both conditions due t lobar shrinkage in collapse and to its being pushed up by bdominal fat in the case of the squat heart but the shadow is essenti ally different.

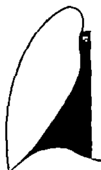


FIG. 85. Lower lobe atelectasis, Stage II.

Moreover there are further film and phy ical findings which are conclusi Collapse as we noted must bring emphysema in this case it is in th costo-phrenic angle, where w can see marked loss of normal lung markings on the film while in the uppe and mid zones we find the essel-markings splayed out much more radially than in the normal. There will theref re be marked tympany in the axilla, off-setting the increasing dullness in the lower inner third posteriorly and w shall hear the blowing breath sounds from the bronchus ove its point f entry near the

are usually slower in development and therefore approximate to the slower shrinkage seen in bronchial carcinoma.

The characteristic shadow of the preceding pneumonia has been described and the changes in broncho-pneumonia which goes on to lobular and lobar collapse have been detailed. In pneumonia there has usually been considerable progress towards recovery with clearance of the homogeneous density from the crisis in temperature before the complication occurs. When collapse sets in by blockage of the main bronchus by a plug of sputum the film goes through a series of alterations similar to those seen in both post-operative pneumonia and bronchial carcinoma that is density begins again at the hilum.

It is essential to remember that the same picture can result from abscess and from tuberculosis at the apex of the lobe (see Fig. 17). Radiography can fail altogether to give the true cause of what is seen. It may have been possible to follow the developing collapse from a rounded opacity close to the hilum but usually one sees only a partial collapse in the first X ray the apex of the lung being already hidden in the hilar shadow. Full clinical and bacteriological investigation and serial X rays must be considered in every case.

Three stages of lower lobe collapse may be detailed: early, half and complete collapse. All three have the homogeneous type of shadow, that is it may alter in density but not in smoothness throughout its extent, no part of it but only the whole standing out on distant viewing as the concurrent emphysema is outside the boundary of the affected lobe. All three stages have the same upper point to their loss of translucency. This lies at the upper limit of the ventricular or auricular shadow on the cardiac border; left and right sided cases are similar in this apical hilar limit to their triangular shape.

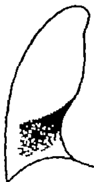


FIG. 84. Lower lobe atelectasis, Stage I.

The first stage shows a loss of translucency diminishing from this point downwards and outwards to the periphery, the upper border being made by the interlobar fissure. It will be realized from the anatomy of the lobe that this means that its apex has already undergone a considerable fall through shrinkage of the triangular lingula, and this we would expect as the lingula being

The third tage is shown on the film as ery dens rea heavie than that of the cardiac bord rather lik a llapsing toy balloon with its neck ttached t th upper cardiac borde On the right side its outer edge is usu-lly concurrent with the right cardiac border on the post ro-anterio film while on th left sid t lies within it It is i such cases in particula that th complementary emph sema may be the only indic tion f abno mality By this tage ther is emphysema on the opposit as w ll as on the affected id and this has an important bear g n physical findings We ha learnt to l that th medi astinum i one ontinuous whole not tructure w th

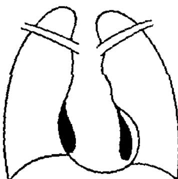


Fig. [illegible]. Lower lobe atelectasis, Stage III.

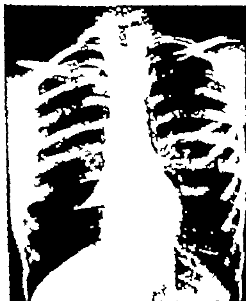


Fig. [illegible]. Third stage [illegible]

eighth or ninth dorsal vertebra because the collapsed lung is acting as a sounding board



FIG. 86 Right lower lobe collapse in second stage.



FIG. 87 Squat heart.

this will undo the tendency to swing It has brought the sub atmospheric pressure nearer to atmospheric o bo e it. The muscle tension still remains the pull on the trachea by the un l lapsed parenchyma is still present If theref re we find a pati t who is dull all over th lowe half of his chest both front and back and in the axilla, on the left side and till has a tense left t mo mastoid he cannot have uncomplicated pneumonia because pneumonia does not mo the mediastinum equally he cannc t have left lower lobe pneumo ia with pleural effusio because this would give a right sterno-mastoid sign and so h must ha collapsed left lower lobe with pleural effusion.

right and left sides As one side of the thorax shrinks, therefore the mediastinum drifts as a whole In collapse of the right lower lobe with drift of the mediastinum to the right there is also drift to the right of the left lower lobe which by emphysema attempts to fill the vacated space This it does by occupying the lower anterior mediastinum and in doing so it must perforce overlap the anterior surface of the heart pushing it backwards We have therefore seen two reasons why the heart swings and goes backwards rather than move to the side of the atelectasis during conditions of respiratory rest Herein lies the reason why we get with complete collapse anterior dullness in the affected cardio-phrenic angle we are percussing the heart pressed back to be continuous in note with the much shrunk lower lobe All over the remainder of the lung and particularly in the lower axillary zone we shall find marked tympany

If however we screen the patient or take two pictures one on complete inspiration and the second on complete expiration we shall see the effect of the strengthened intra-pleural pull on the affected side for the heart comes well over with each inspiration This finding combined with a tense sterno-mastoid muscle will aid us in diagnosis but we shall find the latter the better help if the patient has the complication of fluid in the pleural cavity for



FIG. 90. Left lower lobe collapse with fluid—sternomastoid sign positive on left side.

this will undo the tendency t swing It has brought the sub-atmospheric pressure nearer to atmospheric r above it The muscle tension still remains the pull on the trachea by the uncollapsed parenchyma is still present If the ef re we find a patient who is dull all over the lo er half of his chest both front and back and in the xilla on the left sid and still has a tense left st mo mastoid he cannot have uncomplicated pneumonia becaus pneumonia does not move the mediastinum equally he cannot have left lower lobe pneumonia with pleural effusion becaus this would giv a right sterno-mastoid ign and so he must ha a ollapsed l ft low lobe with pleural effusion

for the same reason as it is not of equal density in its rounded area of loss of translucency on the film It is heaviest in its centre as here are the lobules nearest the bronchial branch along which came the aspirated material Outside these are the lobules which have been irritated in their alveolar walls so that they contain catarrhal inflammatory exudate giving the fine loss of translucency of oedema. *The homogeneity of shadow is a diagnostic X ray feature* and we shall see it remains until cavity formation occurs in those cases that go on to blockage of the bronchus We see therefore that it is of the same consistency as the shadow of lobar pneumonia and of the individual shadows of broncho-pn umonia all three having in common material which is filling lobules and replacing their residual air Putting this in another form, the previous lobar shadow of pneumonia is now in a more or less circular localized area the shadows of broncho-pneumonia are now confluent in a larger single area.

Sometimes abscess involves a considerable area showing no special denser central region but only a wide-spread area of homogeneous loss of translucency There is no particular definition such as appears in lobar pneumonia, making out a definite lung segment



FIG. 92 Lung abscess.

At first the area must be silent apart from its complicating pleural rub It will cause no shift of the mediastinum as its material has the same physiological ffect on lobular lastic tissue as the residual air it replaces There w ll be no terno-mastoid sign nor will it be found in the usual following stage of the abscess which evacuates oluntarily This stage is often referred to as a cavitation because the picture on the film is misread there is no real diff rence except in extent between this second tage and the second evacuating stage of a broncho-pneumonic single lobular affection. Naturally the lobules nearest the draining bronchus evacuate first so they make central translucency within a circular homogeneous shadow mad up of those lobules still undrained This ring shadow has no components of organization or collapse It does not alter within tself or tand out against any surrounding emphysema with distant viewing as a tuberculous cavity wall does (see Fig 48) Stethoscopic findings bear this out w bear just the same âles as we heard in broncho-pneumonia in the mid-phase of inspiration indicating lung material entering the bronchus but naturally they re increased in number and coarseness as a bigger circumscribed area is affected

It is necessary to repeat again even if it appears redundant that the usual lung abscess is not cavity as many students seem to visualize the process It is merely to ll intents and purposes a very localized pneumonia filling lobules with exudat as pneumonia does and differing only by the method whereby it was caused in these lobules It distends them as pneumonic did and does not destroy thei lastic tissue unless t cannot get out into the bronchus Most patients evacuat the material v hintarily at a stage when it contains no lastic fibres Moreo er this method of cure never has with t any lubbing of the fingers the septic material has not been dammed back

Should however this evacuating material block th bronchus the way is open to serious consequences from cavitation to bronchiectasis The first hint can often be got by the film examination the exudat now fluid and thin by th proteolytic enzyme of its polymorphs is dammed back and w see level within the diseased area, confined by the circula r o al hape of outlying lobules which are till filled with large mononuclears phagocytes and polymorphs Should this not be evacuated by drainage by conservati or urgical methods then repai and not resolution will take plac in these surrounding lobules They annot find an

exit for their material content and slowly fibroblasts will enter them organization and collapse will follow drag on the bronchus be the inevitable result and bronchiectatic cavitation by advancing sepsis from lobules to bronchus is the final picture The septic material now destroys the parenchyma, and the foul sputum contains elastic fibres There will be an immediate reaction in the mediastinum To all intents and purposes we have one single cavity of bronchiectasis There we saw how sepsis is the bronchus extended through all the coats of the bronchial wall until it reached the parenchyma and produced a cavity in the lung connected with a saccular cavitation of the bronchus This is just the reverse process leading to the same end result The disease can now extend like any individual cavity in primary bronchiectasis

The physical signs are now those of organization fibrosis and atelectatic collapse By destruction of elastic tissue we have drift of the mediastinum with a positive sterno-mastoid finding to the stethoscope we have the crepitations of fibrosis and the coarse metallic râles of bronchial dilatation and its connection with lung tissue are heard throughout the first and mid phases of inspiration These are exactly the same added sounds as we found in advancing bronchiectasis

Clubbing of the fingers of the septic type comes on rapidly with the blockage of the bronchus If evacuation by efficient drainage is carried out the clubbing disappears quite soon; if it does not do so then the observer should be doubtful if the lung condition is really cured Many cases are shown by t mography to be going steadily if slowly forward to the stage of bronchiectasis in spite of apparent cure by operation This is why there are so many so-called recurrences of lung abscess they are not recurrences but continuances of the same initial uncured lesion. We are now dealing with secondary bronchiectasis not a lung abscess

CHAPTER XVII

TUBERCULOSIS UP TO EARLY ADOLESCENCE

WHATEVER be its out of entry to the body by nhalati n ingestion the tubercle bacillus can always reach the lungs When it comes in contact with any mucous surface it is ingested by a phagocyt carried by it through the mucous membrane to enter the lymph spaces and thence is conveyed to the lymphati node or tissue draining this particula area Its next stage on the journey is th venous blood and so the right heart is reached and, through the pulmonary circulation the lung tissue Should it be held up in a small capillary the phagocyte ple ces the wall to enter the surrounding lymphoid tissue and th t f rm a focus of disease I other words whatever be the manifestation of the disease ir the lungs from childhood to old age tuberculosis is primarily lymphati disease Its forms depend on the reaction of lymphoid tissue It lways ttempts to drain along the lymph channels which we saw in our consideration of applied anatomy are part of the protecti yst m together with th lymph spaces and the glands It succeeds in most cases in th infections of childhood type but its ery evidence of quired immunity in ts efforts to confin th infection t localized areas can be the undoing of the infected subject as age advances ince the lymphoid tissue, so hedged about becomes the breeding-ground fo the bacilli and so itself breaks down It is worthy of not that increasing age brings much increase in lymphoid tissue throughout the lungs thus is doubtless in great part an explanation of the widespread ravages f senile phthisis which yet maintains such resistance as to imulat only hronic bronchitis

Although there are no sharp lines f demarcation dividing the disease into forms f different ages it is convenient to describe its further developments und the headings of Infancy and Early Childhood Childhood t Early Adolescence ' and Early Adolescence and Adult Lif as they show fairly defined types of response of the now infected organism to f esh infections

Tuberculosis in Infancy and Early Childhood

The forms of th disease shown in this age-group re miliary

CHAPTER XVIII

ADOLESCENT AND ADULT TUBERCULOSIS

EPITUBERCULOUS lesions are found up to about the fifteenth year of life although most of them occur before the age of twelve Th reafter we come to the manifestations of tuberculosis most commonly met with in general practice Fundamentally they are the same as those of earlier ages in their interplay of infection and immunity that is they are probably due in the most part to reinfections o reawakenings of previous primary infections It is highly possible ho ever that many of them are continuing primary infections in individuals who have not been infected before the age of adolescence Some patients show exudation with no foregoing or with destroyed foregoing immunity others show lessened immunity and thus varying amounts of consequent fibrosis Some th refore show the spread of the blood-borne type of miliary tuberculosis some show this f rm combined with fibrosis in the individual scattered lesions of chronic miliary tuberculosis hile others show break into a bronchus and give broncho pneumonic or pneumonic types Most however show the disease commencing as far as we know by thei diagnosis through X ray films in those special broncho-pulmonary segments of dorsal branches of bronchi which we studied under applied *anatomy* and saw *involved in the commoner sites for formation* of lung abscess Consid rable fibrosis is the usual response so that fibro-caseous disease and a tendency to consequent cavity formation is the finding in the majority of patients We do not know if such usual adult manifestations are really a secondary or tertiary stage of the disease because we have no mass X ray observations under the age of seventeen or eighteen Similarly we do not know what really happens to a primary infection in adolescence or adult life Recent experience s ggests that a young adult *who is negati e to tuberculin and* gets first contact with an open case of tuberculosis shows film findings exactly lik those which can accompany an rythema nodosum There is a large confused hilar shadow often with scattered indiscrete pulmonary shadows These latter may be areas of lymphangitis and

consequent collapse f bunches f alveoli Som are complicated by pleural effusion It is not uncommon to recover tubercle bacill from the stomach washings Th X ray such as bown in Fig 95



FIG. 95. The X-ray appearances hich can accompany erythema nodosum.

is usually read as Sarcoidosis of lung Th writ has f llowed some such cases by serial films through stag f fleeting shadow in ne both apices t d first nfraclavicular depos t with positi e sputum within ighteen mo ths f th appearanc f erythema nodosum.

In the invol ement of broncho-pulmonary egments the ttack is again through the lymphati system there is no chang i the fundamental pathogenesis f the disease Ou tud es on th anatomical relations f the al coll eminded us that th y connect with the lymphatics through lymph spaces wh ch surround th lobules It i into thes spaces that the phagocyt conta ning the tubercle bacilli now enter after being caught in the lymphatics which accompany th dorsal bronchioles and th first eaction is catarrhal exudat in th al eoli from irritatio f th ir walls This is a non-specific inflammatory oedema th same reaction which we saw in epituberculosis

is not uncommon to find the first recognizable foci of adult tubercle as rounded areas of homogeneous loss of translucency in the inner or outer thirds of the right infra-clavicular region They are round because as we have noted before the cone of infected parenchyma is photographed through its long axis They may be in small and multiple areas up to about ¼ inch in size when first noted but are usually single and about three times this size They are then known as Assmann s foci They are then fairly well defined, and entirely homogeneous The apparent striations on them are really the normal markings of lung tissue *lying anterior to them* on the postero-anterior film as can be shown by taking either an antero-posterior film which *presents them nearer to the surface* and therefore to the film or by doing a tomograph (see Fig 46)

There is one *reaction to the focus that is quite common in the* upper lobe especially the right and is often called erroneously '*tuberculous pneumonia We saw that in epituberculosis there is a possibility that lymphatics round a bronchus can be so swollen by reaction that they occlude the lumen and produce a* localized area of collapse as a complication, A comparable reaction can take place *in response to an acute Assmann s focus Swelling* in the lymph-spaces together with much catarrhal exudate in *irritated alveoli round them can give a heavy shadow part of which* is due to actual lobular collapse The interlobar fissure is drawn up and the *mediastinum well displaced to the affected side.* The lymphatic inflammation is quickly passed to the interlobar fissure *which is always heaviest in its shadow just below the lesion* This is the condition that was compared for differential diagnosis with upper lobe *atelectasis (see Fig 76)*

Rest and serial films will often show the true state of affairs Just as the catarrhal *reaction often disappears round foci in other* parts of the parenchyma now both the catarrh and the lymph angitis can be seen on *serial films to retrogress to more and* more translucency while in the rounded area of the causative deposit we not *infrequently see a rapidly forming* cavity with a fluid level The condition has been therefore but a manifestation of the severity of the attack

The temperature and constitutional distu bance may simulate pneumonia at onset *but the dullness* of the upper lobe is more posterior than anterio and we shall find signs of mediastinal displacement *which* as we have seen can occur only in complic ations of pneumococcal pneumonia Later the film can simulate

abscess formation in a lobe collapsed by bronchial carcinoma but as the cavitation develops in the tuberculous case the rest of the lobe clears above the lesser fissure whereas in true carcinomatous atelectasis the surrounding density remains By this time sputum examinations will generally clinch the diagnosis

The large rounded form of the Assmann's focus is we have seen by no means a necessity Individual acini may be seen on the film as tiny individual dot like foci congregated together and giving no evidence of inflammatory exudate We hav already noted that such single or multiple foci can be concealed by ov rlapping bony shadows Where there is any doubt the rib intersections must be studied very closely and an antero-posterior view taken All this means is that there is less acut invasion or that we are looking at later stage when the xudate has gone it does not mean that these areas will not go on to the same after-effects Serial films show they can proceed to cavitation f slower onset although they are on the whole more inclined to go on t organization and fibrosis without much drag on the bronchus If however they caseate and affect by extension other acini in their immediat neighbourhood they are no d fferent in their film findings from those Assmann foci which appear to break up into their constituent parts and produce picture of small triangular areas that merge into the shadow of the inflamed lymphatics round congested bronchi tracking towards the hilum The disease is following its constant pathogenesis it is attempting to drain by the lymphatics to the hilar glands

This type of individual acinar deposit is common in the supra clavicular apex and means that the posterior apical branch of the upper lobe bronchus has been affected (see Fig 3) Exper ienc with mass radiography shows this is far commoner site of disease than was formerly believed and many cases that show activ Assmann s foci below the collar bone have evidence of previous true apical deposits Indeed this is so common that it appears probable that infra-clavicular tubercle is a second o perhaps a secondary attack to previous and unsuspected more peripheral disease never quite healed Whether uch tru apical tubercle is an outpost of the primary focus of hildhood that remains dormant until adolescenc w may by the modern possibilities of serial film examina ion soon be able to state

It can be extremely difficult to decide whether such dep ‹ are present in the supra-clavicular apex Ov rlapp

shadows the shadow of the sterno-mastoid pleural tenting and pleural striæ can all give confusing shadows The antero-posterior view is often of great help in the final opinion

If we now looked at the cut surface of the post mortem specimen we should see a collection of tiny nodules of collapsed and organized acini each round a central more or less occluded, bronchiole. We might also recognize many white strands due to fibrosis of the peribronchial and perivascular connective tissue the latter narrowing the blood vessels and helping to contribute to the endarteritis obliterans which is a *constant feature of the disease* The hilar glands are hardly if at all affected the fibrosis has allowed but few bacilli to reach them

It is little wonder that tuberculosis is without symptoms or gross physical signs especially to stethoscope for a considerable *time after its establishment in adult form* *It does produce* lack of movement however and very soon afterwards destruction of elastic tissue begins by organization and the results of bronchial blockage The sterno-mastoid sign reflects drag on the mediastinum, but only careful stethoscopic listening will demonstrate the first sound of the *material evacuating from the lobules into their* supplying bronchioles We shall then hear evidence of the bronchiole contents in fine sibili at the very end of inspiration increasing in intensity to reach a crescendo at the very end. Remembering our stethoscope findings in bronchitis we recognize a distinctive and diagnostic difference in the position in inspiration of this sound which is exactly the same in its quality to the ear in both diseases In bronchitis we heard it at the beginning of inspiration, *lessening with th increasing* depth of breathing, and connoting obstructive material consisting of breaking-down elements of the wall what we hear in phthisis is material evacuating into the terminal distal ends of the bronchioles from their connecting *lobules* With these sibili or coarser rhonchi we shall soon hear the dry crepitations of fibrosis also most marked at the end of inspiration and not disappearing with cough; these are the *signs of the peri* bronchial fibrosis In other words

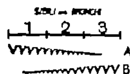


FIG. 98.
A. Bronchitis.
B. Pulmonary tuberculosis

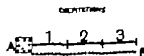


FIG 99
A. With act of cough in pleural thickening.
B. With fibrosis of lung.

we have stethoscopic evidence of infiltration and fibrosis

Thickening f the pleura overlying the broncho-pulmonary segment follows very soon Its stethoscopic sign w hall also find, in a burst of fine dry crepitations with the act of cough which disturbs the fine adhesions of organizing exudate between the visceral and parietal layers

The progress of a focus that keeps its rounded form for some time shows by serial films that it may although rarely seem to disappear altogether It may split as already noted int small individual lesions or go on to a drawn out mass of fibrotic strands and organized areas Sometimes there will be rapid cavitation with occasionally a fluid level at first X ray ecognition. Usually howeve the cavity is due to a slow weakening of the walls which are dragged peripherally in ragged fashion by organ ization here and collapse there so that the film shows an area that has lost in great part its rounded formation and is merging into the shadow

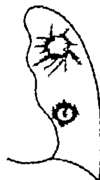
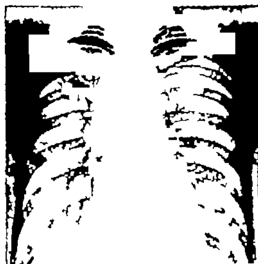


Fig. 100. Cavitation in pulmonary tuberculosis.



of the larger bronchi now outlined by surrounding thickening connective tissue as they track downwards and inwards to the hilum

Critical inspection of the film of an established case with activity gives us a lesson in living pathology If we look at it closely we see it has a background of fine generalized loss of translucency this is thickened plastic pleurisy At the edges of the lesion we see areas of slightly greater loss of translucency they are due to the catarrhal exudate in irritated lobules in the process of invasion *i e* the areas of advancing infiltration merging into the heavier shadows of lobules now containing tubercle bacilli that have penetrated their walls and are producing caseation More centrally we see still denser individual lesions clear cut standing out on distant viewing like the thickened bronchial walls because they have round them lobules distended by complementary mphysema. These denser areas of collapsed and organized lobules are fused and solid in clumps round the dilated and destroyed bronchial wall to form the heavy but broken up outline of cavity The picture is distinctive and diagnostic unlike any other in chest disease

There is possible confusion with the cavity of true lung abscess but, as we have already seen the cavity surround in this case looks

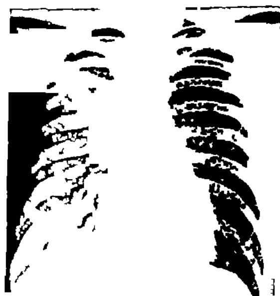


FIG. 101 Same case as in Fig. 102. A-P view show cavity is present.

as if all the affected lobules had been suddenly frozen in situ to form a continuous band with no areas of intervening lobular emphysema and therefore not altering essentially in their detail on distant as against close viewing. We saw also that a further and important point in such differential d agnosis is that the chronic tuberculous cavity is seldom the only evidence of phthisis it is much more often in a lobe throughout which can be seen scattered areas of infiltration with fibrosis



Fig. 103. Tuberculous cavitation.

The advent of the cavity gi es its wn ad entitious sounds The presence or absence of whispering pectoriloquy is not a reliable diagnostic finding th more the co tents of the cav ty at the time of listening the less the pectoriloquy A much more constant sign is the presence of coarse metallic râles They are i quality exactly as those we heard in bronchiectasis and in lung abscess but they are now heard beginning in the second half of inspiration and become more and more insistent with the depth of breathing until they reach a crescendo at the ery end

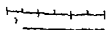


Fig. 104 Metallic râles in tuberculous cavitation.

We must remind ourselves that considerable portions of lung tissue are involved before added sounds are heard It is a fairly safe rule to conclude that signs heard above the clavicle mean the upper third of the lung is involved and signs below the clavicle mean at least half of the lung is involved An example of the influence of other factors in their production is that where laryngeal tubercle is present we can consider the extent three times that indicated by our stethoscope findings

Acute Exudative Phthisis of Adolescence and Adult Life

This type of pulmonary tuberculosis is fortunately nowadays rare in this country as acquired immunity is widespread We do however occasionally see it as a steadily advancing disease unrestrained by any fibrosis Inflammatory exudate in the lobules is rapidly replaced by caseating material which on evacuation fills the bronchi whole areas breaking down to be coughed up and to leave in the lung fields ragged vacancies which are therefore cavities of entirely different mode of formation from those we have already considered As no barrier is placed in the way of lymphatic drainage tubercle bacilli flood the hilar glands and they in turn go on to rapid caseation

The film therefore shows many areas of translucency among widespread homogeneous densities The translucencies are the cavities often of no clear definition but occasionally looking like ticket punchholes where there has been a sudden evacuation through a bronchiole of rapidly caseated tuberculous material round it Bunches of heavily infiltrated lobules then make up what is only an apparently confining wall Hilar shadows are heavy indistinct and confused made up of the caseating glands surrounded by much swelling in lymphatics and much increased hyperæmia.

Movement of the chest is poor there is generalized loss of note with no offsetting areas of emphysema. The predominant part of the affected zones of the lungs is silent, as residual air is replaced by exudation and caseation but now and again we hear coarse rhonchi caused by the bronchial obstruction These are especially noted being exaggerated and insistent even after cough in areas going on to cavity formation by rapid evacuation We are listening to 'tuberculous bronchitis' which is really a flooding of the bronchi by the evacuating caseated tuberculous deposits

CHAPTER XIX

PLEURISY

There are three main forms of pleurisy: fibrinous or dry, sero-fibrinous, or pleurisy with effusion, and purulent or empyema. There is no hard and fast delimination between these forms, seeing that there is always some slight fluid present with the first, while the second and third really vary only in the amount of pus in their cellular content. All three forms can be caused by the pneumococcus, the streptococcus or the tubercle bacillus.

Dry pleurisy may be primary to the pleura by the presence in its layers of the bacillus of Koch. It is however almost always secondary to pulmonary tuberculosis if it be tuberculous, even if it is actually found in the parietal layer, in which case what has happened is that bacilli from a previous frank effusion have penetrated this layer to its intimal coat. Dry pleurisy is also found with pneumonia and broncho-pneumonia and their suppurative complications, and may be diaphragmatic in site when secondary to abdominal disease, which is commonly sub-phrenic abscess.

Its effect on the membranes depends on its cellular content, the higher the polymorph percentage-count the less being the chances of ultimate and permanent damage. This is why lobar pneumonia, while producing a heavy exudate on the visceral layer, yet leaves little ultimate damage in the vast majority of cases, as its greyish-yellow thick deposit, even should it undergo some organization, will be removed eventually by the proteolytic enzyme of its preponderant polymorph content. On the other hand tuberculosis produces an exudate which contains a high percentage of fibrin as against polymorphs, and so is much more liable to proceed to permanent adhesion between layers of the pleura over parts of the lung affected by phthisis.

We can follow these effects of dry pleurisy on the film. We see much more density from a recent pneumonic than from a recent secondary tuberculous pleurisy, but the pneumonic one followed in serial films will almost always disappear, while the tuberculous one leaves behind a haziness of fine homogeneous

type to act as a background to those lung shadows of infiltration and fibrosis which are the underlying cause of its appearance

There are two exceptions to this rule the first being demonstrated in so-called tenting of the diaphragm We saw already that localized lung affections of pneumonic type are slow of disappearance and incline to healing by repair rather than by resolution as against their more generalized forms The same effect is seen in interlobar pleural infections We have noted earlier that very often a localized tent shaped shadow is seen on a film about the mid point of the diaphragm on one side more often the right than the left

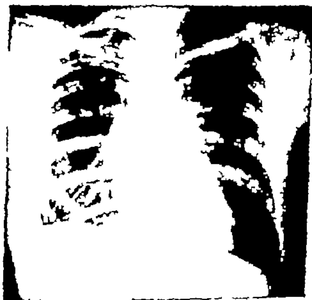


FIG. 105. Filling of costo-phrenic angle plus lung focus; the cause is tuberculous.

We noted the second when discussing the diaphragm It consists in that filling-in of the extreme costo-phrenic angle by a shadow which has no clinical significance unless it be accompanied by a lung focus Without evidence of such a focus it should never be read as tuberculous in origin It is almost always due only to a previous known or unrecognized pleural reaction of pneumonic or broncho-pneumonic origin

The most painful type of dry pleurisy is the pneumonic The progress of the average secondary pleurisy whatever its cause is from viceral to parietal layer each being in olved first by inflammatory reaction and then by the specific reaction of the penetra tion of the causative organism, in the same way as w saw the progression of any lung disease involving the alveoli from catarrhal to specific exudation. The pneumococcus first irritates and then invades the visceral pleura as it reaches the periphery of the lung sector It seldom does more than inflame the parietal lay Œdema stretches and makes this layer supersensitr Dragging by adhesion between the moving visceral and the static parietal layer then produces great pain W th lung abscess pain is not frequent and with plastic pleurisy of tuberculous origin th patient complains more of a rheumatic ache than an acute discomfort unless as in pneumonia he has a widespread reaction to the parietal layer in which case he is almost alway about to develop frank effusion

As already noted sero-fibrinous ffusion diff rs from dry pleurisy only by virtue of the fact that fluid is in amount suffic ent for physical or isual demonstration The cause in most cases seen in general practice is tuberculosis f the lung It is to this latte type that w shall confine ur detailed description f frank pleurisy

The fluid contains much fibrin a high percentag of protein and a preponderance of lymphocytes as against polymorphs when compared with th high polymorph content of the pneumococ l and the rarer streptococcal ffusions It is usually lear and has an opalescence due to suspended fine particles of fibrin asily seen in fluid examined in situ through a thoracoscope and quickly f rming the typical clot in samples withdrawn fo visual and laboratory examination It is not cloudy lik the pneumonic exudat w th its polymorphs tending t form pus I the fibrinous cont nt l es the dangerous liability t the formation of thick coverings on the visceral and parietal layers when the fluid is left to organize n the pleural cavity Tubercle bacilli seldom appear in smears and may fail to appea in culture if the sampl is examined unde fou days from the onset of the ffusion i asion has not y t followed irritation and consequent inflammatory xudation Failure to realize and act on this simple fact of pathogenesis may ha serious c nsequences for the patient by inefficient treatment founded or fait

in one laboratory examination report which has been made too soon and without clinical notes to the pathologist

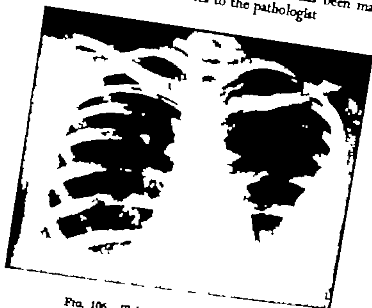


FIG. 106. Tuberculous Pleural Effusion.

To physical examination there will be dullness beginning in the lower axilla and extending upwards with the increase in effusion While there will be silence in the area displacing the lung there will naturally be increased breath sounds over that part of the lung on the limits of the fluid which are by it pressed inwards and upwards the collapsed lung acts as a sounding-board The outstanding point is however the displacement of the mediastinum to the opposite side demonstrated in a positive sterno-mastoid on that side and usually supported by evident displacement of the apex beat to palpation and auscultation

We may remind ourselves how the lung reacts to the presence of fluid in the pleural cavity When the negative intra-pleural pressure is brought nearer to that of the atmosphere there will be less outward pull on the underlying lung Its elasticity will come into play therefore and draw it inwards to the anchorage f all elastic tissue at the hilum From the first the reduction of the pleural pressure on the affected side will make that on the opposite side greater in comparison and so the mediastinum will be first

of all drawn to this opposite side With more fluid th pressure in the pleural cavity will be greater than that of the atmosphere, and so begin to bear in on the parenchyma that is is it greater than the pressure of the residual air in the lobules But w can see that there does not need to be any actual push from the affected side in order to give a positiv sterno-mastoid on the other

After this point the reaction of the lung and the mediastinum depends on whether they are healthy o already diseased A lung with preceding fibrosis will naturally resist collapse and will move bodily against the mediastinum, while a fixed mediastinum *e g* one that has been affected by previous pleurisy w ll esist displacement. What this means is that we must consider all these separate factors and not conclud that the amount of mediastinal displacement is in anything like exact ratio to the amount of fluid o the actual change in pressure in th intra pleural space

While it must be stressed that no pleural ffusion has been fully investigated unless exploratory puncture for visual and laboratory diagnosis and blood film examination, have been carried out it is none the less true that tuberculous exudate has characteristic film finding Unfortunately this cannot be demonstrated by the print of the film as it can by looking at the film itself on th viewing box In its early days it has a specific fine loss of translucency through which the ribs can be seen almost as clearly as they would be with the presence of a pneumothorax T wards the mid zone from above downwards there is a comparative density concave inwards running from the axilla to the diaphragm, but this is not in the uncomplicated case clea singl line of demarcation It is mad by the combined shadow of fluid and lung, the former lying to some depth laterally both in front and behind the latter If there is no under lying lung disease we ought to be able to see exaggeration of the normal vessel markings to the inner ide of this junction, owing to the hyperaemia in the lung contracted towards the hilum If these markings cannot be seen we must suspect consolidation from f r example pneumonia

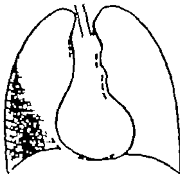


FIG. 107 Tuberculous pleural effusion.

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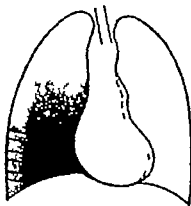


FIG. 108. Pneumonia right lower lobe, with fluid. Note loss of vascular markings, as in uncomplicated pneumonia, but displacement of mediastinum due to complicating effusion.

The picture is therefore not like that of uncomplicated lobar pneumonia of the lower lobe with its density at the base decreasing from the diaphragm upwards towards the lingula and equal at any one level between the axilla and the mediastinum In pleurisy there is increasing loss of translucency from the axilla inwards to the junction of fluid and lung Moreover it will be differentiated also by the position of the heart and the medi astinum for we saw in our study of the pneumonias that they do not cause displacement

If fluid has been present for some time before the film has been taken the conca e density may have assumed a sharp outline This is because organization of the fibrinous exudate on the isceral pleura has already taken place and the patient is thereby in danger of incomplete re-expansion of the lung even with immediate and complete withdrawal of fluid Should the effusion be of still longer standing, organization of the fibrin floccules gives a dense white shadow occupying the area between the axilla and the now sharply defined border of the lung fixed and compressed

The shadow is not now anywise different from that of true empyema although the latter in its acute stage will cause displacement to the opposit side This will be a definite aid in visual diagnosis but it must call for the accessory aids of blood and exploratory-puncture examination The usual cause in general practice cases is the pneumococcus for true tuberculous empyema is a rarity and seldom met with except as a complication of a previous clear effusion especially if such ffusion has occurred as a hydro-pneumothorax

The average case of such idiopathic pleurisy as just described lears completely with no remaining evidence in pleura or lung If we see on the film some filling of the costo-phrenic angle and no lung focus is evident on a postero-anterior film we ought to take lateral and lordotic films and even tomographs if we wish to

be as certain as is humanly possible of our final opinion Often the small tuberculous deposit is in the periphery in the upper mid zone, or in the supra-clavicular apex from in ol ement of the posterior apical bronchus

Interlobar effusions apart from that in the lesser fissure on the right side are seldom evident or demonstrated to their true xtent in the postero-anterior film A lateral shows them l arly forcing apart the interlobar septum and usually bow shaped because they are pressing on the parenchyma of both lobes (see Fig 8) Fluid in the main fissure can gi on the postero-anterior film shadow very like that of tru diaphragmat c ffusion r empyema secondary to abdominal disease g sub-phrenl abscess Both sit over the diaphragm and re nclmed to follow its outline The physical signs however will help us t diff rentiate because while the latter gives us d llness both ant rlorly and post ri rly the dullness due to interloba effusion is especially marked in front A lordotic picture lds us too f r the true interlobar effusion rises through the middle of the lung field lik drawn-out tent sitting on the diaphragm

The usual dry diaphragmatic pleurisy show no idence t film examination to account for the acut should and upper arm pain that accompanies it

Pneumothorax

From what has been said of the effects f pleural effusion on lung elasticity and mediastinal shift w an ualize the changes in pneumothorax without furthe detailing them The ffect on the film findings of the underlying lung is the same and the diagnosis must rest on the lt rations noted both w thin and w thout the lung field As against pleural effusion w now ha complet absence f shadow between the rib interspaces outside the line of the retracted lung no loss f translucenc and no lung markings re evident (see Fig 2)

In the mple or non-tuberculous case t is tremely rare fo fluid to appear in the pleural space and it may be possible to see on the edge of the collapsed lung emphysematous bullae one of which has burst t produce the pneumothorax In the case where pneumothor omplicates an already demonstrable r undemonstrable lung tuberculosis fluid is exceedingl common

The commonest abnormality of mediastinal glands is of course due to tuberculosis and is dealt with in the chapter on Childhood Tuberculosis

Lymphadenoma Hodgkin's Disease

This condition usually appears first on the postero-anterior film as a moderately sized shadow in the upper mediastinum It is slightly more dense than the shadow of the superior vena cava which it generally covers This is because it is almost always first evident

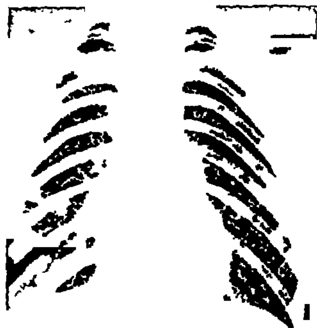


FIG. 109. Lymphadenoma.

in the right para-tracheal glands even if it has been bilateral from the beginning Its outer border is clear-cut but in a series of bulges as against the more continuous sweep of the rounded or oval

outline of other abnormal shadows i the same position It lower border and the greatest abnormal width of the mediastinum lie



FIG. 110. Lymphadenoma Oblique view of case in Fig 95.

above the uppe aortic shadow Serial films show lon increase n siz across the mediastinum thus displacing the trachea and th oesophagus and nto the neck Such spread may obscure the in lvement f othe groups of glands lthough they an sometimes be identified on hard postero-anterio ant ro-posterio o lat ral iew If spread includes the pulmonary tracheo-bronchial and h lar groups the picture is one of overlapping egg l k w llings long an indefinit upper cardiac border W may now see pulmonary invol ement in streak nes spreading from the out border along the lines of the bronchi as definit nodules or as homogeneous irregular shaped patches some w th the density f pneumonia and some perhaps edged with ar as of ground-glass appearance because they re causing collapse f surroundi g alveol

At this stage we may need every accessory aid including irradiation to which it responds less to distinguish this disease from lympho-sarcoma



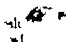
FIG. 111 Lymphosarcoma.

Lympho-sarcoma

This is the next commonest abnormality of glandular origin It is generally bilateral from its onset In early stages its density is comparable to that of lymphadenoma but it is generally more rounded in its right border It grows more rapidly to produce marked broadening of the superior mediastinum and may so obscure the aortic shadow that it cannot be diagnosed against aortic aneurysm

Dermoid

The oval homogeneous ... alt ... by the a erage dermoid of pericardial ... w l ... num on its inner side and gives a ... (See Fig 49)



On the postero-ant rio film it is usually situated lower than the level of the aortl ı h We may requ r an artificial pneumothorax to be certain of its connection with the mediastinum

When d rmoid arises from embryonic remnants of th thymus the blique lew w ll how it lying in the anterio superior media stinum It does not displac the trachea r the oesophagus We hav already noted that film examination lone w ll not diagnose

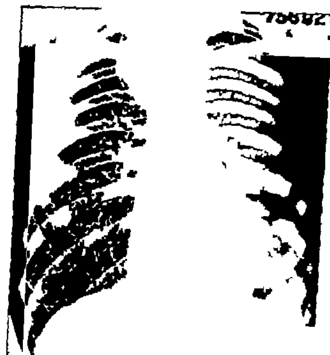


FIG 112. Dermoid of lung

this t rm from large entral neurofibroma from the erage h dat d st the same are Only ery rarely does d rmoid originat from the parenchyma

Neuro-fibroma

This the commonest benign chest *tumour* arises from an intercostal nerve It is usually *discovered by* chance during mass radiography *or screening* Its *loss* of translu ency is comparable



F 113 Neurofibroma.

in homogeneity and density with lymphadenoma but it is mo e rounded in its outer edge Antero-posterior postero-anterior lateral and oblique films w ll demonstrate it as lying in the posterior superior mediastinum tter Bucky film of the spine may show its connection with th nerve It is soft in structure and low in growth and therefore shapes itself against the resistance of surrounding structures hence its usually clear-cut and rounded outer edge on the postero-anterior film It does not displace the trachea

the œsophagus Occasionally it develops in a central lung position when as noted bo pneumothorax may be necessary for differential diagnosis against dermoid and hydatid cyst



Fig. 114 Neurofibroma, lateral iew

Substernal Thyroid

A subst rnal thyroid gi es a triangular homogeneous shadow in the upper anterior mediastinum with the base f the triangl l the thoracic outlet and the apex po ting downwards It may be more pronounced on one ide or be completely to one id of the mid l ne but usually depresses the main essels and pushes the aorta to the left so that the aortic knob i prominent Very often it presses backward on the trachea. Screening will show t rises when the patient swallow

CARCINOMA

Lobar Carcinoma

Other parenchymatous growths have been considered in the chapter on Round Shadows

Primary sarcoma of the lung is rare but primary carcinoma is common It usually begins in a large bronchus and therefore produces lobar collapse first by increasing obstruction and later by actual alveolar involvement in the manner described in the chapter on atelectasis

There we noted its tendency to cast a shadow which is densest at the hilum Hard films may show this density to be *made of two* shadows a very dense on of enlarged glands within another sl ghtly less dense of collapsed lung

Loss of lung volume is shown by the falling-in of the rib-spaces outside the affected area and by the drift of the mediastinum and the heart to the same side We saw why this drift is more marked n lower lobe than in upper lobe atelectasis; not only is there more loss of volume but the heart is much more readily displaced from normal in its *lower and heavier half* The swing from this fixed position of drift still further to the affected side on inspiration and the return on expiration so easily seen on the screen is also much more marked w th lower lobe and compl te lung collapse than with upper lobe collapse Pulmonary fibrosis o pleural blitera tion do not prod this swing We have already seen how we can get further aid in the diagnosis of collapse against fibrosis if we note the positions of the oesophagus and the trachea

Nerve n ol ement is quite common in upper lobe carcinoma as the re urrent laryngeal on the left side and the phrenic nerve on the right side can very easily become embedded and compressed in masses of enlarged glands in the upper mediastinum Extension of a right uppe lobe carcinoma can therefore produce an increasing rise of the right d aphragm until complete paralysis of the nerve brings complete immobility of the diaphragm in the position of complete expiration equivalent to that seen with excision of the nerve The conjunction of a raised diaphragm and an atelectatic right uppe lobe is diagnostic of right upper lobe carcinoma

Screen ng will give us still further aid The movement of the diaphragm is paradoxical This abnormal movement occurs also in lo r lobe carcinoma It cannot be seen on the right side since the shadow of the muscle is merged with the liver shadow

but if the patient is given a Siedlitz powde it is possible to demonstrate it on the left side unless ther is a large pleural ffusion

When collapse of the lower lobe by carc noma has reached what we described as the third complet stage w may requir lipiod l injection for diagnosis against foreign body or bronchiectatic collapse In both these conditions w are lmost ertain to find some lipiodol in the collapsed lobe

Atelectasis f the whol lung oc rs when the growth n a main bronchus W find a homogeneous loss of translucenc all over one side with a density arying from that f pleural ffusion to that of empyema The f llow ng po nt w ll d us to arr at the true diagnosis The rib-spaces w ll be fallen n on the side of the shadow The mediastinum and h rt w ll be d wn t this side and w ing still furthe er with ach inspi at on The œsophagus will lean o er and the trache w ll be d agged w ll



FIG. 115 Carcinoma of the lung—nodular (medial) form.

over The alue of physical examination the differential diagnosis of collapse with omplicating ffusion has bee mentioned

The appearance and differential diagnosis of the type of nodular carcinoma which throws a *single rounded shadow* has been discussed in the chapter on Rounded Shadows Three other main forms of nodular carcinoma remain to be mentioned mediastinal medial and basal The mediastinal or hilar form originates near the bifurcation of the trachea Instead of blocking the lumen it erodes the wall and as it involves the glands and all the mediastinal tissues produces a shadow which is large rounded homogeneous and extremely dense Its edge may be well-defined or ragged If it is ragged it can usually be seen to send out irregular fibrotic like threads of differing size and shape into surrounding lung tissue

The medial form is due to carcinoma of a main-bronchus close to its origin The shadow looks as if it were part of the main cardia shadow resting like a bump on the entricular border Th *outline may be sharp or quite irregula* The shadow changes very l ttle to serial xaminations o er a period of time

The basal form gi es an ill-defined lung shadow attached t the hilum by many irregular streaks It is usually of varying densities as it has within it areas of obstruction of bronchioles Very often it proceeds to the usual picture of lower lobe complete collapse

INDEX